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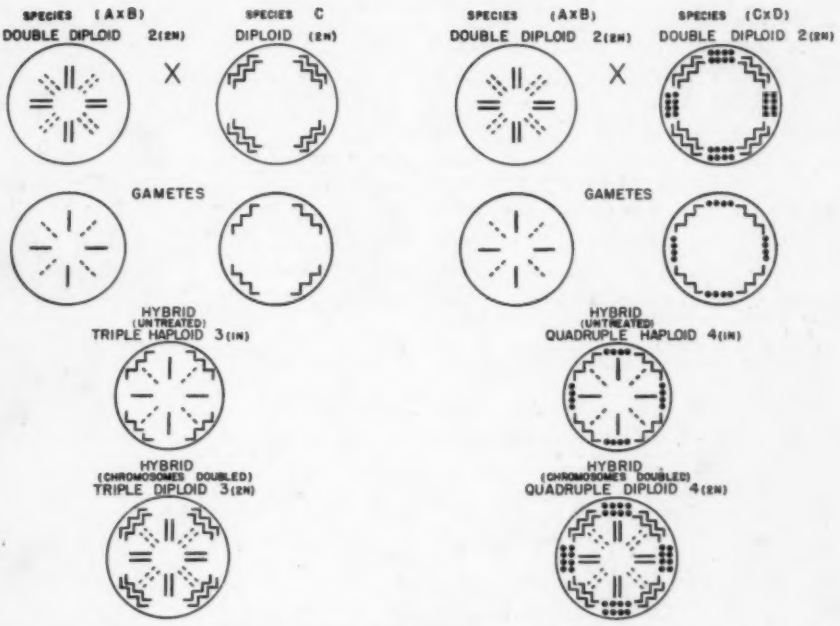

A PEDIGREE OF ARTIFICIAL POLYPLOIDS

(SEE PAGE 421)

Color Mosaics in Pigeons
Genetics of Coat Color in Horses
The "Moo-Cow"—An Alleged Hybrid
The Jackson Memorial Laboratory
Gene Symbols for Cotton Eugenics
A New Linkage in the Mouse
Inherited Hernia in Dogs
Origin of Hairless Cats

VOLUME 30 OCTOBER, 1939 NUMBER 10

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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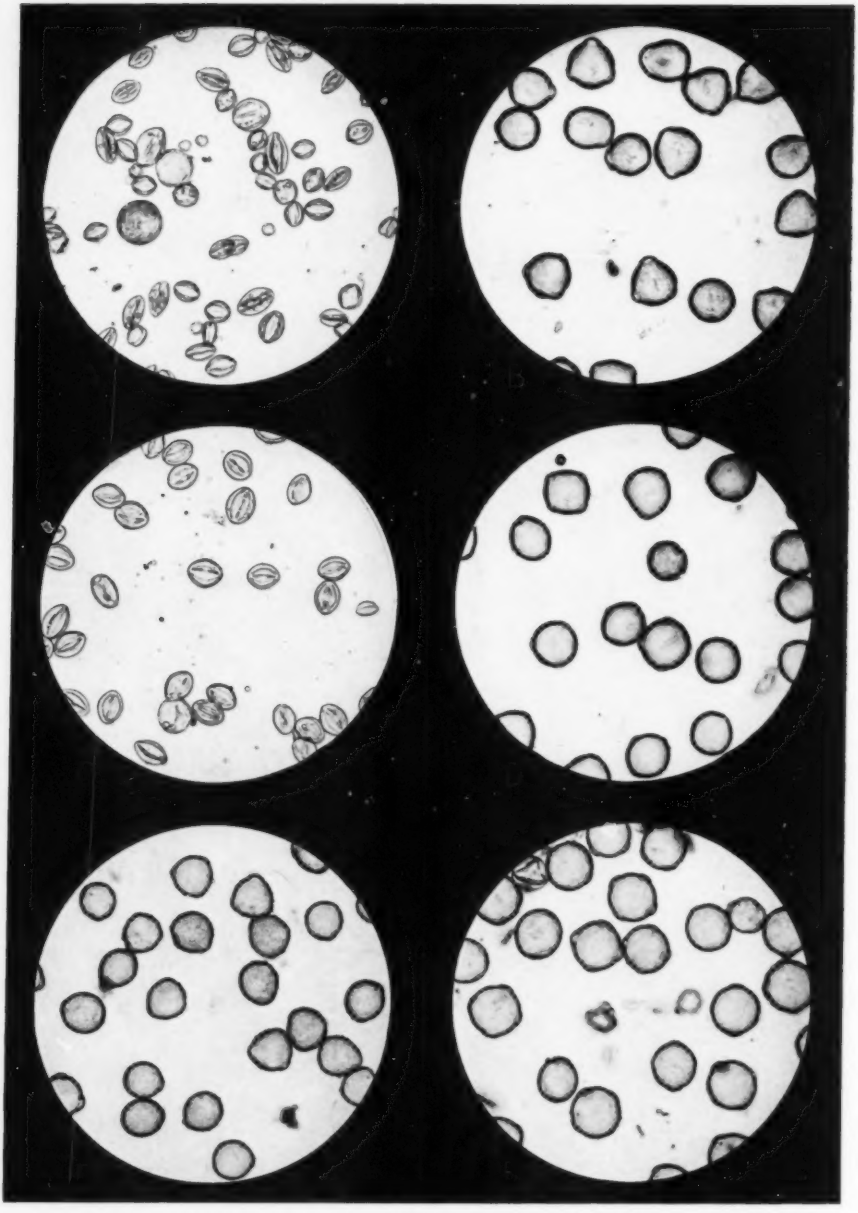

POLLEN CHANGES DUE TO DOUBLING CHROMOSOME NUMBERS

Frontispiece

A and *C* show aborted pollen of two sterile species hybrids; *B* and *D* show good pollen resulting when these hybrids are made fertile by colchicine treatment. *A* and *B* are from the hybrid *Nicotiana glutinosa* × *N. sylvestris; C* and *D* are from the hybrid *N. tabacum* × *N. glutinosa.* In such hybrids, which have aborted pollen grains, the presence of flowers with good pollen is adequate proof that the chromosome number has been doubled. *E* and *F* show the pollen changes in *Nicotiana sanderae.* At left (*E*) are pollen grains from a 2*n* plant; at right (*F*) those from a colchicine-induced 4*n* plant. The pollen grains from the 4*n* plant are approximately twice the volume (1.25 times the diameter) of those from the 2*n* plant. Note, also, the four germ pores characteristic of grains from the tetraploid in contrast to the three germ pores of grains from the diploid. These differences in pollen size and shape are presumptive evidence that the chromosome number has been doubled (× 175).

INDUCTION OF SIMPLE AND MULTIPLE POLYPLOIDY IN NICOTIANA BY COLCHICINE TREATMENT*

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IN an article some time ago in the JOURNAL OF HEREDITY,[4] *Nicotiana* was listed as one of the plants which showed evidence of having had its chromosomes doubled in number through treatment with colchicine. The evidence consisted in characteristic thickening and roughening of the leaves, changes which have come to be recognized as being associated with induced tetraploidy.

Through the kindness of Dr. F. O. Holmes, seeds to produce the sterile F_1 hybrid *N. tabacum* × *N. glutinosa* were made available for experimentation. The intention was to render these hybrids fertile by doubling the number of their chromosomes and thus producing double diploids (allotetraploids, amphidiploids). It had earlier been pointed out[2,4] that probably one of the most valuable applications of a method of doubling chromosome number at will would be the manufacture of double diploids. It was with considerable disappointment, therefore, that the seedlings of these F_1 hybrids were found to die soon after the stage shown in Figure 7 of the article just cited from the JOURNAL OF HEREDITY. At about this time a number of geneticists reported failure in their attempts to double chromosome number in Nicotianas through colchicine treatment and thus called in question the general applicability of the colchicine technique. It seemed desirable to accept the challenge and to make a more careful study of the reasons for our earlier failures with the genus.

It soon became apparent that while we could grow Jimson Weeds successfully, we did not know how to grow tobaccos. Our F_1 *Nicotiana* hybrid seedlings died because their roots became badly infested with nematodes and a fungus root rot. By growing them in sterilized soil we overcame this difficulty and were able to raise a high percentage of the plants to maturity. We also overcame the root troubles by grafting onto Jimson Weed stock. Two such grafted scions of the hybrid *Nicotiana tabacum* × *N. glutinosa*, along with numerous other plants, were sprayed with an emulsion containing colchicine and induced to double their chromosomes and to produce fertile pollen. This emulsion was made according to the following formula:†

Emulsion Formula

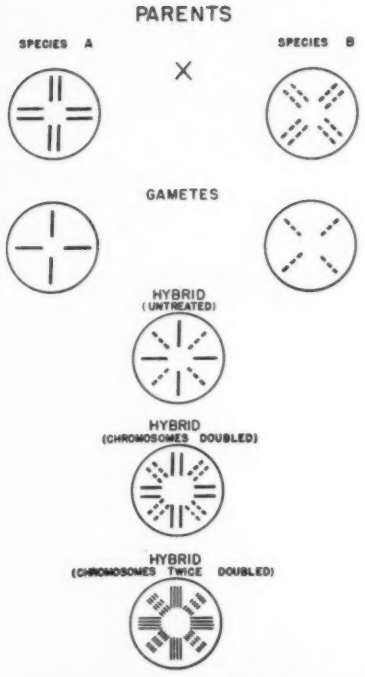
stearic acid	1.5 gms.
morpholine	0.53 c.c.
tap water	20.0 c.c.
lanolin	8.0 gms.

Add the water to the stearic acid and morpholine and heat the mixture until the stearic acid is melted. Then stir to a creamy soap solution. Add the lanolin and continue heating until the lanolin is melted and the mixture just below the boiling point. Now stir until a thick, creamy emulsion results, and continue stirring intermittently until the emulsion has cooled to room temperature.

This emulsion is thick and must be

*The present article is a report of a paper presented in September, 1938, at the Woods Hole meeting of the Genetics Society of America, of which an abstract has been published in *Collecting Net.*[24] The investigation has been supported in part by a grant from the Carnegie Corporation to the Carnegie Institution of Washington.

†Formula and morpholine were obtained from Carbide and Carbon Chemical Corporation, 30 East 42nd Street, New York City.



NO. OF CHROMOSOME SETS	NO. OF CHROMOSOMES IN EACH SET	DESIGNATION	FERTILITY
4	2	DIPLOID (2n)	FERTILE
4	1	HAPLOID (1n)	
8	1	DOUBLE HAPLOID 2(1n)	STERILE
8	2	DOUBLE DIPLOID 2(2n)	FERTILE
8	4	DOUBLE TETRAPLOID 2(4n)	FERTILE

PRODUCTION OF DOUBLE DIPLOIDS AND DOUBLE TETRAPLOIDS

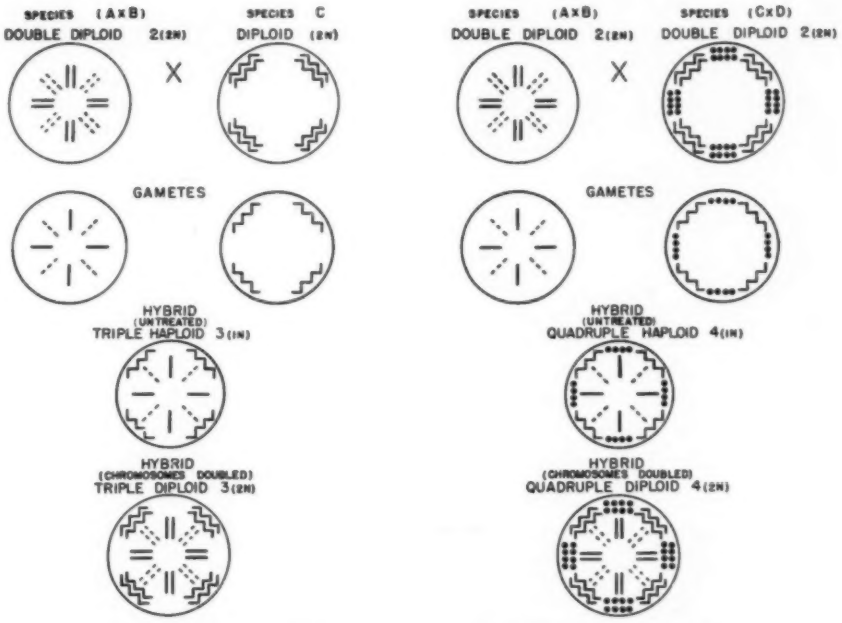
Figure 1

Above is shown diagrammatically the chromosomal changes involved in the synthesis of fertile species hybrids. Double diploids (allotetraploids of some authors) have been obtained through colchicine treatment, and experiments are in progress to produce double tetraploids.

diluted for spraying. We have diluted it by adding to one part of thick emulsion, four parts by volume of water. Colchicine is then added to any desired concentration; we have found 0.4 per cent to be a convenient strength, although various other concentrations between 0.1 per cent and 1.0 per cent colchicine have been used. The resulting emulsion is sufficiently fluid to be sprayed without difficulty, and will remain stable over long periods of time if kept in a cool place.

This dilute emulsion (containing 0.4% colchicine) was sprayed on the new *Nicotiana* shoots twice weekly until growth was retarded. Usually not more than three or four applications were necessary to check growth. Such treatment was found to be more effective in doubling chromosome numbers than spraying twice daily for 3-4 weeks with an 0.8 per cent aqueous solution of colchicine. Too prolonged or intense treatment may kill the buds that have been sprayed and force distant dormant buds to grow out into *2n* branches. Weak treatment may show no evidence of chromosome doubling. The proper treatment will check the buds which may later be forced to grow out into *4n* branches, if the *2n* buds are kept from growing.

Our experience with one species and two hybrids of *Nicotiana* reported in the present paper, and other recent work,[9,14,15,23] would indicate that doubling chromosome numbers in this genus should offer no great difficulties to the



PRODUCTION OF TRIPLE AND QUADRUPLE DIPLOIDS

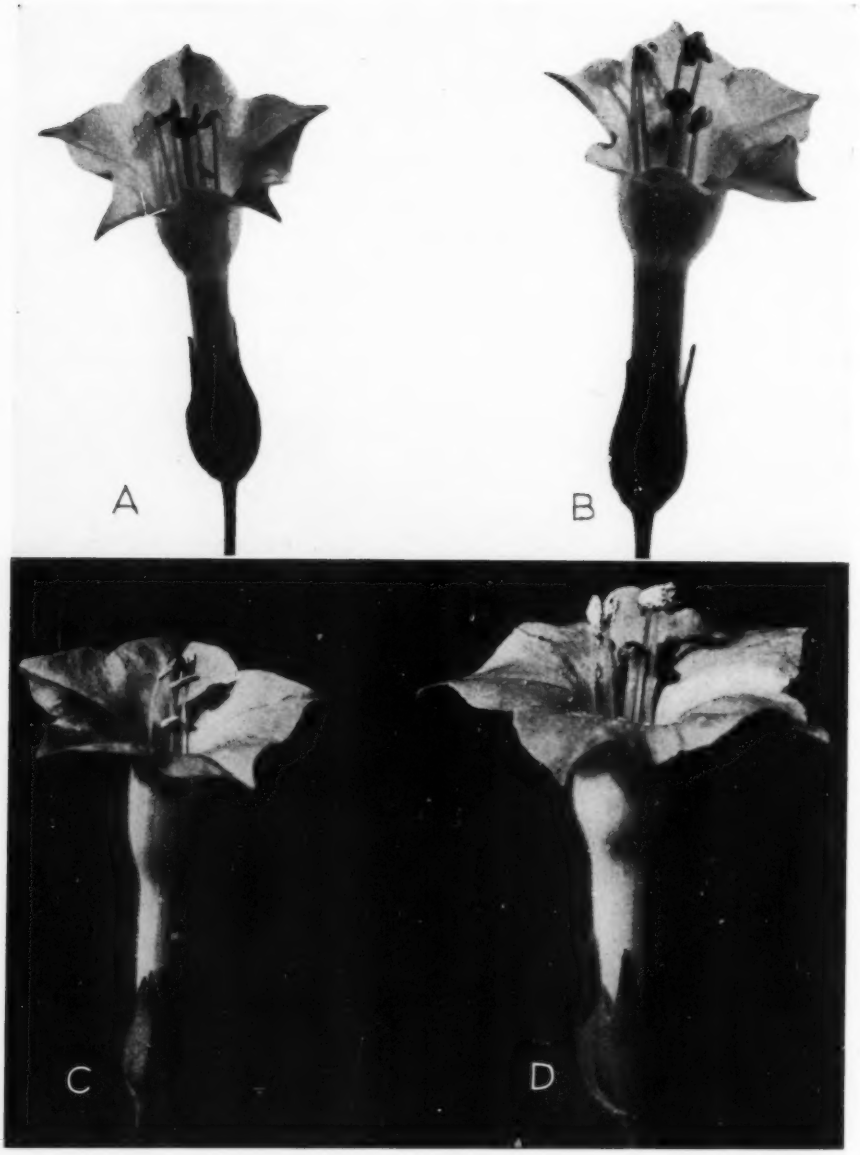
Figure 2

The above diagram shows changes through hybridization and subsequent chromosome doubling suggested for the synthesis of more complex pure-breeding hybrid forms. A triple diploid is reported in the accompanying article, and a quadruple diploid is being synthesized.

experimenter who is familiar with the cultural conditions required and who has patience and a reasonable amount of skill in adapting the type of treatment to the responses of the particular form under investigation.

Hybrids are readily formed between species of *Nicotiana* and many of these species combinations are sterile. There seems to be no better group, therefore, in which to test the effect of doubling chromosome number in sterile species hybrids. A series of diagrams which have been used in a general discussion of polyploid problems[3,6] may help in understanding how the sterile species hybrids of *Nicotiana* may be transformed into pure-breeding races. The chromosomes of hybrids which are completely sterile fail to mate to form pairs at the reduction divisions. Without chromosome mating there can be no sexual reproduction. Hence the sterility. What prevents mating of chromosomes is not known, but it is reasonable to assume that the incompatibility between chromosomes in certain species combinations is connected in some way with the degree of their evolutionary relationship.

In Figure 1 the chromosomes of species *A* are represented by solid lines, those of species *B* by dotted lines. Each species is assumed to have four different sets or kinds of chromosomes. In the sporophyte ($2n$) there are two chromosomes in each set. In the gametophyte and gametes the number is reduced to half and each set contains only one chromosome. This stage is called haploid ($1n$). In the hybrid between species *A* and *B* the chromosomes are assumed to be unable to pair, with re-



DOUBLE HAPLOIDS STERILE; DOUBLE DIPLOIDS FERTILE

Figure 3

A, blossom from double haploid (sterile hybrid) *Nicotiana tabacum* × *N. glutinosa* (36 chromosomes); *B*, blossom from double diploid plant (fertile) of same hybrid (72 chromosomes); *C*, blossom from untreated hybrid *N. glutinosa* × *N. sylvestris* (24 chromosomes); *D*, blossom from fertile plant of same hybrid as *C* (48 chromosomes). Doubling the chromosome number induces fertility, increases length and thickness of filaments and thickness of style, corolla tube and flower stalk. In the blossoms on left the pollen is aborted; in the blossoms on right the anthers are filled with good pollen (×1½).

sultant sterility of pollen and egg cells. The sterile hybrid has only one chromosome in each set and should therefore be called a haploid ($1n$). There are now, however, eight sets or as many sets as in both the two parents combined. Such a sterile hybrid, therefore, may be called a double haploid—$2(1n)$. By the use of colchicine the chromosomes may be doubled. The resulting type will be diploid ($2n$) since it has two chromosomes in each of the eight sets. It will be fertile since each of the chromosomes of both species *A* and *B* will have duplicates from their own species with which to pair. Since the number of sets is the sum of those in the two diploid parents it may be called a double diploid—$2(2n)$. If the number of chromosomes were again doubled there would be four chromosomes in each set and the plant should be called a double tetraploid—$2(4n)$. The word tetraploid means four-fold and would seem applicable only to cases in which each kind of chromosome is represented four times. The term amphidiploid, as a Latin equivalent of our double diploid, describes what actually happened to the chromosomes. Allotetraploid is an unfortunate term since it emphasizes the mere change in total number without regard to how this change has been brought about. In a terminology regarding changes in chromosome number we believe it will give a clearer picture of what happens if we focus our attention upon the different kinds or sets of chromosomes the numbers of which are doubled, tripled, or quadrupled to form $2n$, $3n$, or $4n$ types.

In Figure 2 is shown by diagrams how it should be possible, with our knowledge of chromosome incompatibilities within the genus, to build up triple and quadruple diploid Nicotianas.

Induction of Double Diploidy

Several hundred seeds from the cross *N. tabacum* (n=24) × *N. glutinosa* (n=12) and two plants of the hybrid *N. glutinosa* (n=12) × *N. sylvestris* (n=12) were secured from Dr. F. O. Holmes of the Rockefeller Institute at Princeton. A portion of the seed was planted untreated, as a control, and another portion was treated for different lengths of time in different concentrations of colchicine in water.

Seed Treatment

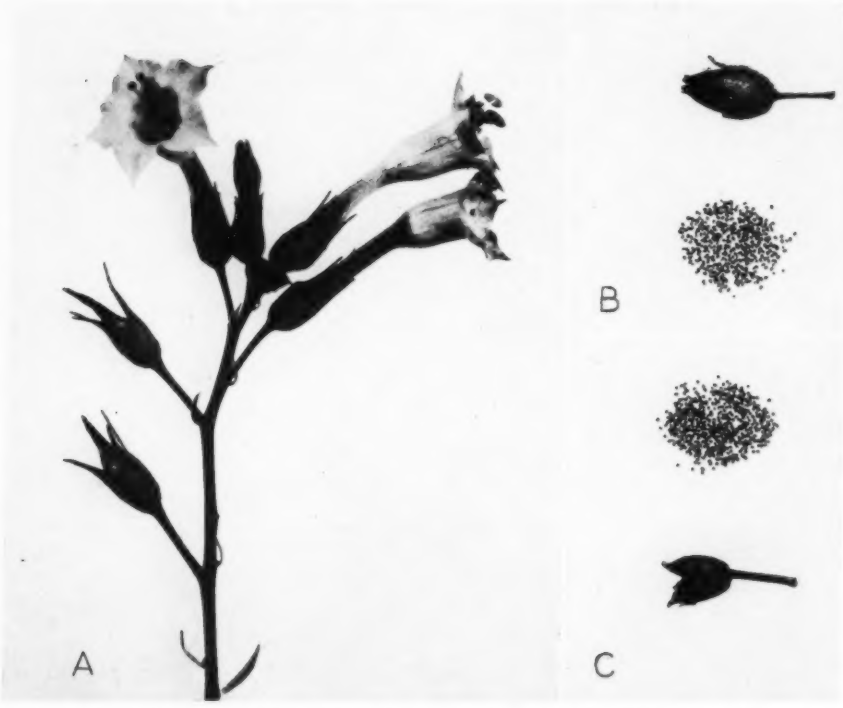
Forty-one control plants were secured from the untreated seeds of the hybrid *N. tabacum* × *N. glutinosa*. These plants all proved to be highly infertile, with 98 per cent of their pollen grains aborted, as shown in Frontispiece *C*.

It has been shown that very little meiotic pairing takes place between the chromosomes of *tabacum* and *glutinosa*.[7,8,17] Segregation is irregular, and the pollen grains and egg cells are largely aborted. We have found these hybrids to be completely sterile; none of our 40-odd untreated plants have formed seed or set capsules.

In the hybrid whose chromosomes have been doubled, however, two identical chromosomes of each kind are present. Pairing takes place between these homologous pairs,[17] segregation is relatively normal, and pollen is well formed (Frontispiece *D*). Good pollen, therefore, is a simple and positive test for chromosome doubling in this case. In Table I the results of treating seeds of the cross *N. tabacum* × *N. glutinosa* are recorded.

The longer period of treatment (2 days) proved more effective with a 0.4 per cent concentration, or even with a 0.2 per cent concentration than the shorter time (1 day) at 0.8 per cent. In the most effective treatment (0.4 per cent colchicine for 2 days) double diploidy was induced in 6 plants out of 13, or in over 46 per cent of the cases. All ten plants made fertile by this method have set capsules with viable seeds (Fig. 4*A*, *B*).

Microscopic examination of the pollen, in most cases, is really not necessary, for the flowers on the fertile plants, as Clausen[7] has shown, are quite distinct from those on the sterile ones. The stigma is larger in diameter, the filaments are thicker and longer, the anthers are heavily laden with pollen, and the corolla tubes and flower stalks are



CAPSULES AND SEEDS FROM COLCHICINE-INDUCED DOUBLE DIPLOIDS

Figure 4

A, branch on sterile hybrid, *N. tabacum* × *N. glutinosa* which bears fertile flowers and capsules after treatment with colchicine; *B*, detail of mature capsule and seeds of same hybrid; *C*, capsule and seeds from another hybrid, *N. glutinosa* × *N. sylvestris* which had been made fertile by colchicine treatment. Doubling the chromosome number makes chromosome pairing possible; as a result, good gametes and seeds are formed. Seeds from both hybrids have shown a high percentage of germination (× 2/3).

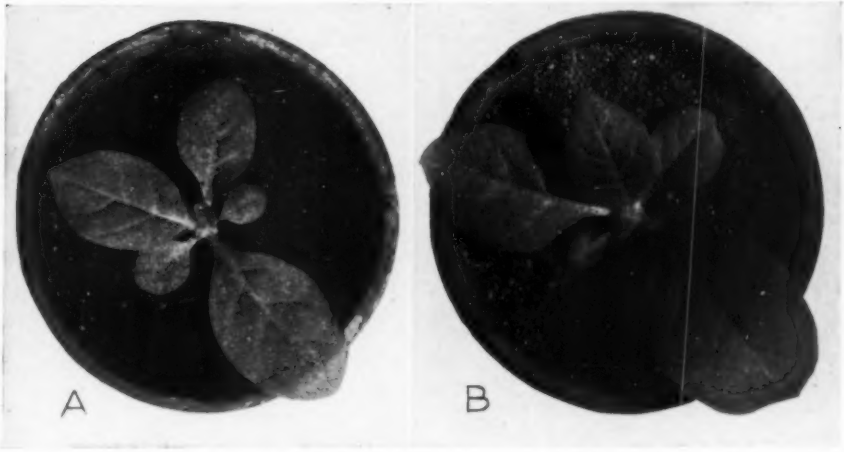
somewhat thicker (Figure 3*B*) as compared with the sterile ones (Figure 3*A*).

Figure 5*A* shows a sterile, untreated seedling of this hybrid; while Figure 5*B* is a second generation double diploid seedling of about the same age. The two are readily separable because of the broader and thicker leaves of the latter, even at this early stage. The two plants also differ in stomata size. The stomata cells from the lower epidermis of a mature leaf of the sterile hybrid average 32 microns in length; those from a similar leaf of a double diploid plant average 40 microns in length.

Root tips of the fertile seedlings have been sectioned and found to contain ±72 chromosomes. This is the number to be expected when doubling occurs in this hybrid, which possesses 36 chromosomes (24 from *tabacum* and 12 from *glutinosa*).

Emulsion Spray Method

Of the 41 control plants of the hybrid, *tabacum* × *glutinosa*, which had flowered and shown themselves to be sterile, 39 were cut back severely. The new branches sent out were sprayed with the emulsion whose formula was given ear-



STERILE AND FERTILE HYBRIDS DISTINGUISHABLE AS SEEDLINGS

Figure 5

A is a seedling of the sterile hybrid *N. tabacum* × *N. glutinosa,* with 36 chromosomes. *B* is a second generation fertile seedling derived from the same cross, with 72 chromosomes. Doubling the chromosome number not only renders this hybrid fertile, but also makes its leaves broader (× 4/5).

lier in this paper. Of the plants so treated, five, or 12.8 per cent, have produced branches with varying amounts of good pollen (10 per cent to 90 per cent) and most of these latter have set capsules and formed good seeds.

The two available plants of the other hybrid, *N. glutinosa* × *sylvestris,* were sterile and formed pollen with 90-95 per cent aborted grains (*A,* frontispiece). Several cuttings of these plants were made and sprayed with the emulsion described above. Later, one of these produced a branch bearing flowers with 95 per cent good pollen (*B,* frontispiece) and set capsules with many seeds (Figure 4*C*). These seeds proved very fertile and produced several score of vigorous seedlings (Figure 6*A*) with 48 chromosomes (two complements from *glutinosa* and two complements from *sylvestris*). The leaf stomata of these double diploid plants average 39 microns in length, while the stomata from the double haploid parent average only 26 microns. Figure 3*C* shows a blossom from an untreated sterile plant of the hybrid *glutinosa* × *sylvestris.* Figure 3*D* is from a second generation fertile double diploid of the same hybrid. Note the increased flower size and abundant pollen of the latter.

Fertile races of both hybrids are being carried on. Unfortunately, the same difficulty with root-rot is being experienced as was encountered with the sterile hybrid parents.

In addition, seeds from the fertile plants of both hybrids have been treated with colchicine in an attempt to again double the chromosome number and produce double tetraploids, 2(4*n*), or allo-octoploids, as Greenleaf[12] has termed them. Figure 6*B* shows a seedling of the double diploid race of *glutinosa* × *sylvestris* that has been treated by the emulsion spray method. It has rough, thick leaves that indicate tissue in which the chromosome number has again been doubled. If the doubling has included the germinal tissue, this plant should give rise to offspring with 96 somatic chromosomes (4 of each kind) and should represent the double tetraploid, 2(4*n*), of Figure 1.

The synthesis of a complex hybrid

containing *glutinosa* (n=12), *sylvestris* (n = 12), *glauca* (n = 12), and *langsdorffii* (n=9) is in progress. Plants of the double diploid, *glutinosa*×*sylvestris* are now on hand, and seeds of the hybrid *glauca*×*longsdorffii* have been treated with colchicine in order to render it fertile. If successful, we should then have available the two double diploids, *glutinosa*×*sylvestris* and *glauca*×*langsdorffii*. By crossing these and treating the seeds with colchicine, we should obtain the quadruple diploid illustrated in Figure 2.

Tetraploidy

Seeds of the crimson garden tobacco, *N. sanderae*, were also treated with colchicine, in order to double its chromosome number. *N. sanderae* is a normal diploid (n=9) with bivalent association in meiosis; successful treatment with colchicine leads to autotetraploidy, with resultant formation of quadrivalents.

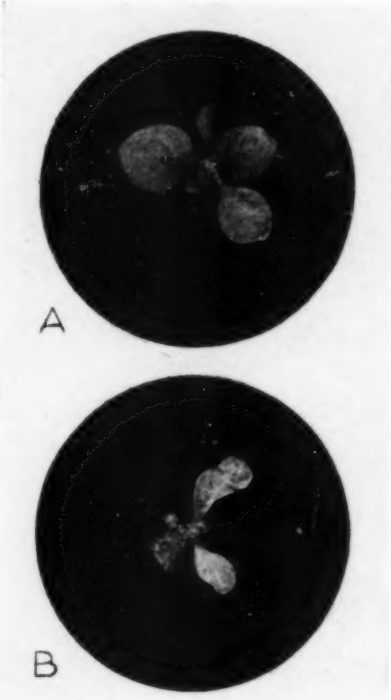
Table II summarizes the results of treatment of this species.

Pollen of the diploid (1n grains) can easily be distinguished from that of a tetraploid (2n grains). The 2n grains (Frontispiece *F*) have twice the volume, and consequently are about 1.25 times greater in diameter than the 1n grains (Frontispiece *E*). In addition, the 2n grains usually have four germ pores in contrast to three germ pores in the 1n grains.

A large portion of the treated plants fall naturally into two classes: those with flowers having all 1n grains, and those having a high percentage (75-100 per cent) 2n grains. There are some exceptions, however; some plants have from 10 to 65 per cent 2n grains, the others being 1n. It seems likely this admixture of pollen sizes in one plant, or even in a single flower, is due to the plant's being a chimera with germinal tissue partly 2n and partly 4n. In Table II this group has been arbitrarily divided; those with 50 per cent or more 2n grains were placed with the tetraploids, and those with less than 50 per cent 2n grains with the diploids.

Tetraploid flowers were tagged and crossed with tetraploid flowers from other plants (*N. sanderae* being largely self-sterile). Seeds from such crosses were planted along with diploid controls. Cytological examination of root tips showed the offspring of controls to be diploid (Figure 10*A*), and the offspring of the treated plants to be tetraploid (Figure 10*C*).

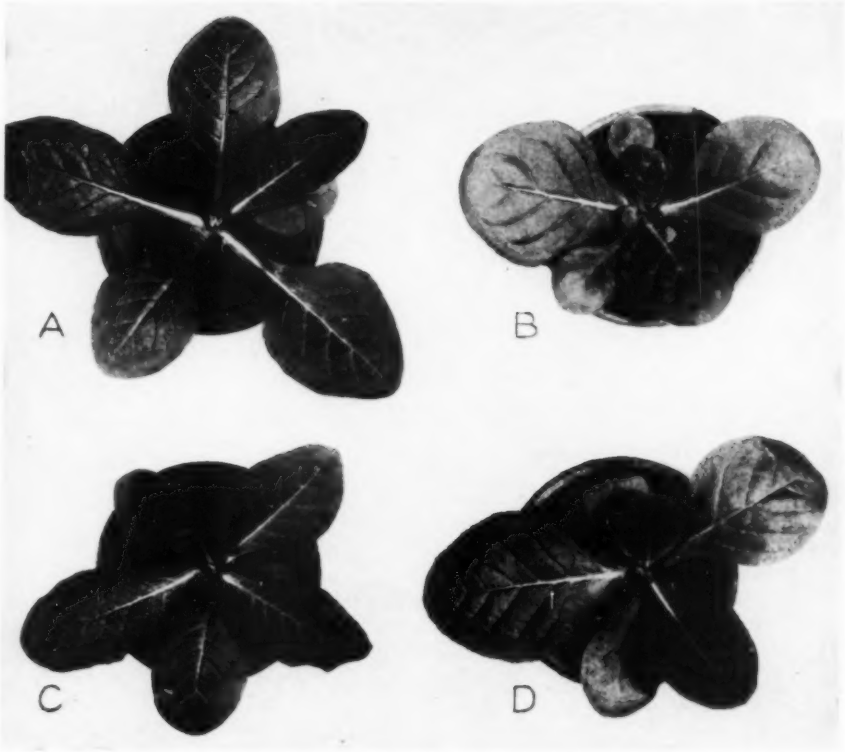
The young second generation tetra-



PRODUCING A DOUBLE TETRAPLOID

Figure 6

A, double diploid, 2 (2n), seedling obtained from the double haploid, 2 (1n), *N. glutinosa* (n=12) × *N. sylvestris* (n=12) by doubling the chromosome number with colchicine. This seedling has two *glutinosa* and two *sylvestris* complements of chromosomes and is therefore fertile. *B*, double diploid seedling which is being treated with colchicine. The rough and deformed leaves indicate that a second chromosome doubling has been accomplished in some of the tissues. These now have four chromosomes of each kind and can therefore be called double tetraploid, 2 (4n). (× 4/5).



DIPLOID AND TETRAPLOID SEEDLINGS

Figure 7

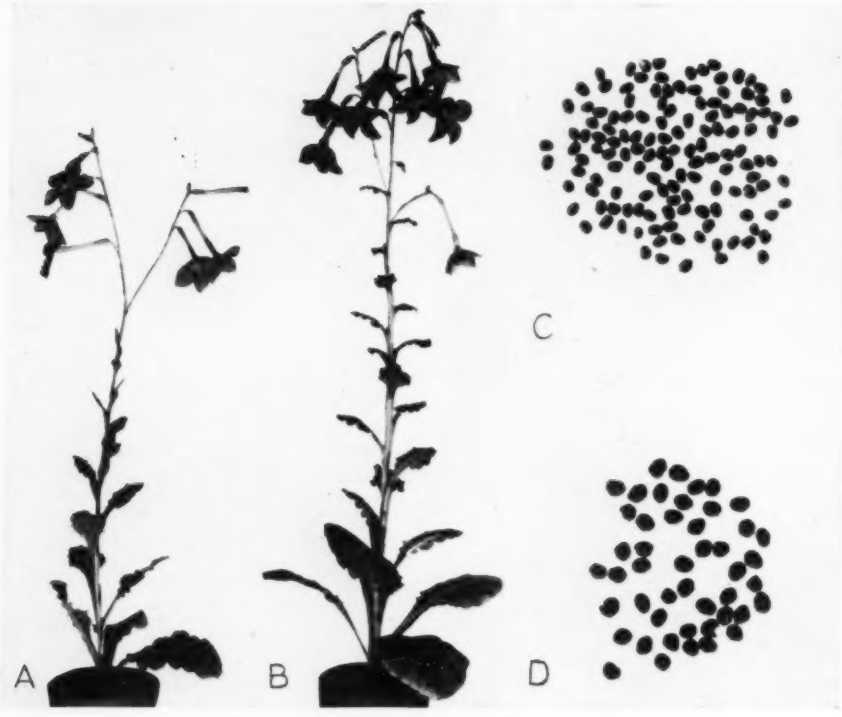
Young diploid seedling plants of *Nicotiana sanderae* (*A*, *C*) differ measurably from comparable induced tetraploids (*B*, *D*). These are second generation tetraploids, being seedlings from diploid plants treated with colchicine to induce chromosome doubling (× 2/5).

ploid plants have thicker and broader leaves (Figure 7*B* and *D*), with shorter petioles than the diploids (Figure 7*A* and *C*). The leaves are also thicker and somewhat more rigid. The average length of stomata cells in full-grown 2*n* leaves is 33 microns, as compared with 47 microns for the leaves from the 4*n* plants.

Mature plants of the diploid and tetraploid races grown under similar conditions in the greenhouse are shown in figure 8*A* and *B*. The tetraploid is larger, has a thicker stem, and is generally more robust than the diploid. The tetraploid blossoms tend to be larger, have broader petals and thicker corolla tubes.

Figure 8*C* and *D* show seeds harvested from diploid and tetraploid plants, respectively. Increased seed size has proved an unfailing criterion of tetraploidy.[5, 6]

Tetraploid plants were crossed with diploids and triploid individuals have been obtained (Figure 10*B*). Of four triploid plants examined cytologically, two were good 3*n* plants with 27 chromosomes, but two were deficient for chromosomes. These latter had 25 and 26 chromosomes respectively. The four tetraploid plants examined all had 36 chromosomes.



4*n* PLANTS AND SEED LARGER THAN 2*n*

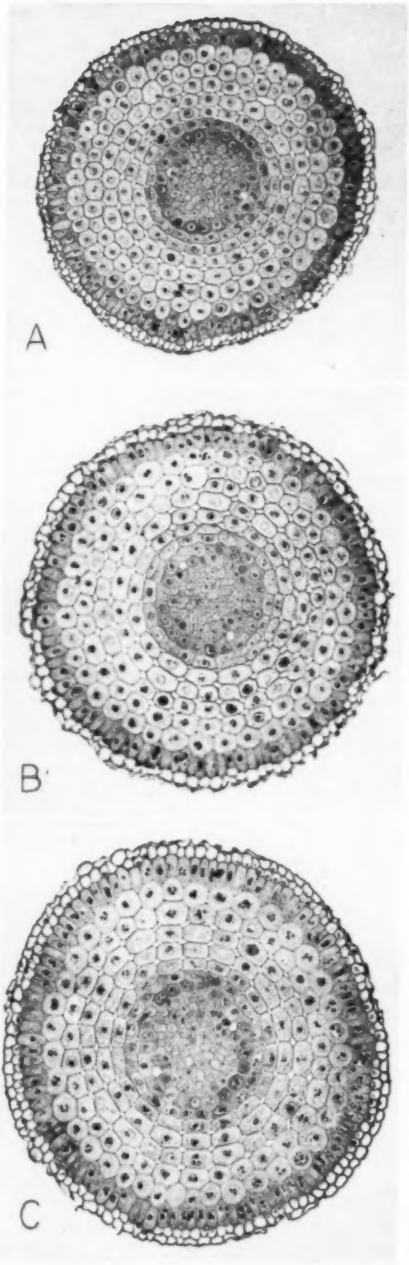
Figure 8

A, 2*n*, and *B*, second generation 4*n* plants of *N. sanderae*. These plants are the same age and were grown under similar conditions in the greenhouse. The 4*n* is markedly more robust than the 2*n* (× 1/8). *C*, seeds from 2*n* plant (× 3). *D*, seeds from 4*n* plant (× 3). Increase in seed size has been found to follow chromosome doubling.

Entire cross sections of 2*n*, 3*n*, and 4*n* roots are shown in Figure 9*A*, *B*, *C*, respectively. It is extremely difficult to choose representative roots from a polyploid series like this one, because there is often a wide variation in the diameter of roots from a single plant. The largest root of 12-15 available from each of plants with 18, 27, and 36 chromosomes was selected. The sections are through the same level in each case (90-100 8-micron sections back from tip of root). They probably give a general picture of the structure of these three types of roots, but the series has not been studied statistically.

The similarities in organization of these sections are striking: the number and arrangement of cells in the central stelar area is about the same; the number of cell layers along a radius of the cortex is ±6 in all three cases; each section has a single row of columnar epidermal cells, and about two rows of dead root cap cells surrounding the epidermis. They are definitely built after the same pattern.

The differences this series exhibits are quantitative in nature rather than qualitative. The diploid is approximately 0.39 mm in diameter, the triploid 0.43 mm, and the tetraploid 0.46 mm, showing a gradual increase in diameter with increase in chromosome numbers. The



total number of cells is approximately the same in each section (possibly a few more in the $2n$). There is thus an increase in average cross-sectional area of cells as the series is ascended. This is in accord with an earlier investigation[22] on flower stalks of polyploid Daturas.

Triploid and tetraploid seeds have been treated with colchicine in an effort to produce hexaploid and octoploid flowers, as has been done in *Datura*.[3] So far, three of the treated $4n$ seeds have produced rough plants with $8n$ blossoms, as determined by pollen measurements. From the treated $3n$ seed, one rough plant with a high proportion of good pollen has been obtained. This plant apparently has $6n$ germinal tissue. None of these higher polyploids has set seed as yet, although several self- and cross-pollinations have been made.

Discussion

A fertile race of the hybrid *glutinosa*×*tabacum* has long been known. It was described as "digluta" in 1925[5] and 1928[7] by Clausen and Goodspeed. The frequency of the spontaneous occurrence of fertile plants in this hybrid is very low; a fertile plant having been reported only the one time, as far as we can ascertain. The frequency of fertile plants in our most effective treatment was 6 in 13 or in over 46 per cent of the cases. This is doubtless many thousands of times higher than would be expected spontaneously.

So far as we are aware, a fertile race of the other hybrid dealt with in this paper (*glutinosa*×*sylvestris*) has not previously been reported.

According to previous investigators,[10, 13] the species *tabacum* (n=24) may be considered a natural double di-

STRUCTURE OF $2n$, $3n$ AND $4n$ ROOTS

Figure 9

Cross sections taken at the same level through comparable roots of diploid (*A*), triploid (*B*), and tetraploid (*C*) plants of *N. sanderae*. The roots of this series exhibit striking resemblance in organization, but differ quantitatively. There is an increase in diameter of roots and in average cell area as the chromosome number is increased (× 125).

CHROMOSOMES OF 2*n*, 3*n*, AND 4*n* PLANTS

Figure 10

Somatic chromosomes from root tips of second generation plants of *N. sanderae*. *A*, diploid (2*n*=18); *B*, triploid (3*n*=27), and *C*, tetraploid (4*n*=36) (× 2000).

ploid of the hybrid *sylvestris* (*n*=12)× *tomentosa* (*n*=12) or *sylvestris*×*tomentosiformis* (*n*=12). If this be the case, the fertile race of *tabacum* (*n*=24)×*glutinosa* (*n*=12), discussed in this paper, is really more complex than a simple double diploid, 2(2*n*), and might properly be considered a triple diploid 3(2*n*), (Figure 2). However, since the origin of *tabacum* was a natural process, and is not completely understood, we have chosen to consider it simply as a diploid throughout the present paper.

Recently, Greenleaf[11, 12] has produced double diploids and double tetraploids from two other *Nicotiana* hybrids, (*sylvestris* × *tomentosa* and *sylvestris* × *tomentosiformis*) by treating decapitated plants with a paste containing heteroauxin. Callus tissue forms on the cut surface, and from this callus tissue adventitious buds arise. He has recorded 13.7 per cent of such buds with doubled chromosome number. His method seems rapid and relatively simple and may have advantages over the colchicine method in the genus *Nicotiana*, which responds readily to auxin treatment.

Kostoff, [14, 15] using colchicine and acenaphthene, has produced *Nicotiana* double diploids of the following hybrids: *alata* × *sanderae*, *suaveolens* × *alata*, *excelsior*×*velutina*, a double tetraploid of the cross *alata*×*sanderae*, and autotetraploids of *rustica*, *sanderae*, and *glauca*. The characters of these polyploid forms agree closely with the characters of those described in the present paper.

At the Richmond meetings, Smith[23] reported having produced three *Nicotiana* amphidiploids (double diploids) by colchicine treatment: *tabacum*×*glauca*, *tabacum* × *sylvestris* and *glutinosa* ×*glauca*. He induced autotetraploidy in *N. tabacum*, and *N. rustica* by the same treatment. His double diploids produced good pollen and viable seeds. Contrary to our findings for the species, *N. sanderae*, he reports his autotetraploid *tabacum* and *rustica* to be smaller, with smaller, thicker leaves, and to have roots smaller in diameter than the diploids.

The tetraploid race of *N. sanderae* we have developed differs from the diploid much as have previously reported tetraploids.[18, 15] It has broader leaves, thicker stems, larger seeds, larger pollen grains, larger roots, larger stomata, larger flowers, and in general, is more robust. From a horticultural point of view it appears more desirable than the diploid from which it arose.

We have found that our sterile hybrids could be made to produce 10-20 per cent good pollen by merely spraying the buds with colchicine during the time of meiosis in the anthers. Such flowers,

however, rarely if ever form seed. Since meiosis does not occur simultaneously in anther and ovary, it seems likely that both egg cells and pollen are usually not affected by such treatment, and that the gametophytes with undoubled chromosome number fail to function in such cases.

The fertility produced by spraying, reported earlier in this paper, was brought about by treating very young branch tips, before flower primordia were in evidence. As a result, a number of vegetative sectors were formed which were entirely double diploid. These bore flowers which formed good ovules as well as good pollen, and set seed.

It is of interest that in our experiments no seed-treated hybrid from the cross *tabacum*×*glutinosa* bore both sterile and fertile flowers. If the treatment was severe enough, the entire plant became fertile; if the treatment was weak, the plants remained sterile. In other species it is not unusual to find only a portion of the plant affected, possibly only a single branch or flower, after seed treatment with colchicine.[4] It is also to be remembered that both fertile and sterile sectors occurred regularly on a single plant of this same hybrid when young shoots were treated by the spray method. These facts may indicate that the region of the embryo that gives rise to the germinal tissue in this hybrid is confined to one or a very few cells; or possibly, that the early divisions in this region are more or less synchronous (since colchicine effects only dividing cells).

Evidence from stomata size, however, seems to indicate that other tissues of the hybrid plant need not have their chromosome number doubled, even though all the germinal cells have been so affected.

It had been shown previously[20, 21] that tetraploidy is usually accompanied by an increase in stomata size and a decrease in stomata frequency. With this in mind, we undertook a study of the stomata of the seed-treated hybrids, in order to separate double diploid plants from sterile ones before flowering time, if possible. The stomata lengths enabled us to divide the plants into two groups: those with stomata like the untreated, sterile plants (average about 32 microns in length) and those with larger stomata (average about 40 microns in length).

After flowering had occurred, stomata size was correlated with actual fertility, or sterility, and complete agreement was not found. All untreated plants tested were sterile and had small stomata. Of the 17 unsuccessfully treated plants tested (those remaining sterile) 14 had small stomata, like the controls; but 3, although with undoubled germinal tissue, had large stomata, characteristic of the double diploid. Of the 10 fertile plants produced, 6 had correspondingly large stomata, while 4 had stomata characteristic of sterile plants.

Since the germinal and epidermal tissues normally arise from different embryonic layers, it seems that this inconsistency may be explained by assuming that the layer giving rise to the epidermis in some cases is doubled, while the one giving rise to the germinal tissue remains unaffected, or vice versa, producing a periclinal chimera for chromosome number.

No actual chromosome counts have yet been made in the tissues in question; so this explanation must remain tentative for the present.

Summary

Through colchicine treatment, chromosome numbers have been doubled in the single species, *Nicotiana sanderae* (n=9) and in the sterile species hybrids, *N. tabacum* (n=24)×*N. glutinosa* (n=12) and *N. glutinosa* (n=12)×*N. sylvestris* (n=12). Seed treatment gave as high as 100 per cent tetraploids in *N. sanderae* and as high as 46 per cent plants with doubled chromosome number in *N. tabacum*×*N. glutinosa*. An emulsion spray containing colchicine (for which formula is given) is more effective than a water solution in treating shoots. Of 39 plants thus sprayed, over 12 per cent gave branches with doubled chromosome numbers.

The term double haploid, $2(1n)$, is suggested to describe a species hybrid which is sterile on account of chromo-

some incompatibility. Doubling the chromosome number of a double haploid would produce a double diploid, $2(2n)$. A second doubling would produce a double tetraploid, $2(4n)$.

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Among the papers which have appeared since the present article was submitted should be mentioned the paper on *Nicotianas* by H. H. Smith in the July issue of the Journal of Heredity.

TABLE I. Results of treating the double haploid, Nicotiana tabacum x N. glutinosa, in the seed stage, with colchicine solutions.

Treatment		No. plants reaching maturity	No. plants with sterile pollen	No. plants with good pollen	Percentage plants with good pollen
Time	Concentration				
1 day	H_2O(control)	41	41	0	0
1 day	0.4%	7	6	1	14.3
1 day	0.8%	15	14	1	6.7
2 days	0.2%	7	5	2	28.6
2 days	0.4%	13	7	6	46.2

TABLE II. Results of treating seeds of the diploid, N. sanderae, in solutions of colchicine.

Treatment		No. plants reaching maturity	Number 2n plants	Number 4n plants	Percentage 4n plants
Time	Concentration				
1 day	H_2O(control)	13	13	0	0
1 day	0.1%	12	9	3	25
1 day	0.2%	10	6	4	40
1 day	0.4%	6	3	3	50
1 day	0.6%	5	0	5	100
2 days	0.05%	6	6	0	0
2 days	0.1%	4	2	2	50
2 days	0.2%	6	3	3	50

HEREDITARY UMBILICAL HERNIA IN DOGS

JAMES McI. PHILLIPS and THOMAS M. FELTON

VARIOUS types of hernia are quite common in man and in domestic animals both in the larger varieties of economic value,[1] and in the small pet stock.[2] A summary of the literature concerning its etiology in the various zoological species by Warren and Atkeson[3] shows a number of theories as to its causes, but the general concensus of opinion seems to be that either the abnormality itself or a structural weakness causing a predisposition to hernia is heritable.

The different forms of hernia seem to be due to specific factors. Warwick[4] concluded that two recessive factors, acting together, produce inguinal (including scrotal) hernia in male hogs, while female hogs of the same constitution remain normal, and that the evidence at hand indicates "that it is largely if not entirely independent of umbilical hernia in its incidence."

Wriedt,[5] in discussing hernia in swine, states: "and I have data showing that there is a dominant factor causing umbilical hernia. Other facts indicate that there are also recessive factors causing umbilical hernia."

Warren and Atkeson found that umbilical hernia, independent of other varieties of rupture, occurred in Holstein-Friesian cattle, and concluded that "The defect seems to be sex-linked. . . . Hernia seems to be a dominant character in males but in females the mode of inheritance is questionable." and "The limited data indicate that hernia is inherited in a simple Mendelian fashion and may be due to one of a pair of autosomes carrying a sex-limited factor for hernia."

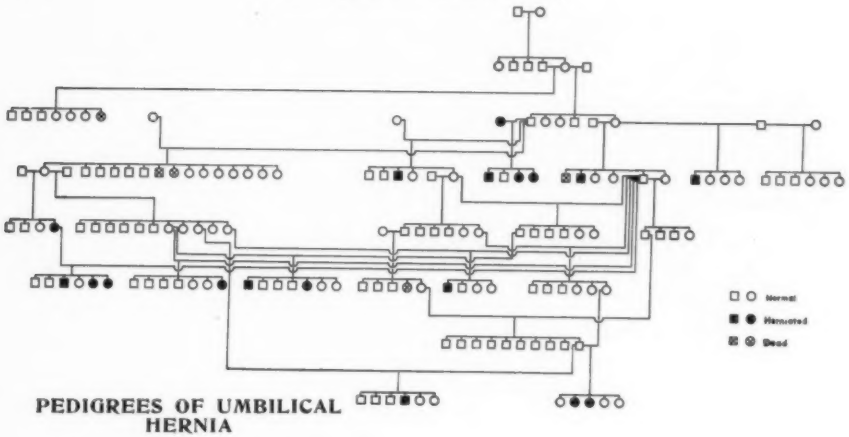
Canine umbilical hernia appears within the first five weeks after birth, and seems to be due to a failure of the normal occlusion of the umbilical ring, so that as intra-abdominal pressure increases with the growth of the pup, omentum and intestine are forced through its aperture, pushing ahead of them the peritoneum covered by skin, thus producing a hernial sack containing one or both of these structures. The hernias are usually comparatively small, the largest we have seen in an adult dog being no larger than a walnut. Sometimes the rupture disappears spontaneously shortly after weaning, and it is often proportionately smaller in adults than it was during puppyhood.

In collies, cocker spaniels, and bull terriers we have observed familial umbilical hernia not associated with any other variety. No records had been kept in the collie kennel and the mortality in new born pups was so high that accurate analysis was impossible. The breeding stock consisted of one normal dog, one normal bitch, and four herniated bitches. When the dams had hernia approximately one half of the pups of both sexes which survived after birth were affected; but the normal bitch's puppies were sound. The dog and two of the affected bitches were sable and white and the other two bitches were tricolors.

A cocker kennel which kept accurate records enabled us to chart the incidence of umbilical hernia through a period of twelve years. Ten females and eight males were affected. Of these one male was black, three were red, one was red and white, and three were black and tan. Two of the bitches were black, five were red, two were tricolors, and one was red and white.

Two matings of herniated by normal gave four normal and six herniated pups. Nine matings of normal to normal gave thirty-four normal pups to eleven affected. A very large number of normal to normal matings were negative for hernia. There were no matings of herniated to herniated in this commercial kennel.

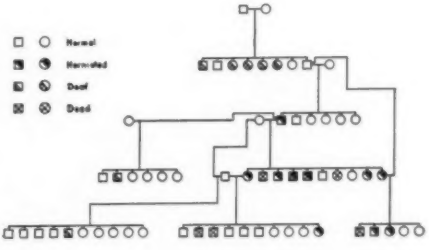
No scrotal or inguinal hernias occurred in any of the dogs under observation, but one cocker with a congenital



PEDIGREES OF UMBILICAL HERNIA

Figure 11

A pedigree extending over twenty years of Cocker Spaniel breeding shows ten females and eight males affected. The distribution of hernias is so irregular that multiple recessive factors are probably involved.



HERNIA AMONG BULL TERRIERS

Figure 12

This strain has been selectively bred for white through many generations. This accounts for the large number of deaf individuals, as deafness and white, or extreme white spotting are known to be associated in several breeds of dogs.

abdominal hernia on the left side near the edge of the ribs was mated to a dog known to be heterozygous for umbilical hernia and a litter of seven normal puppies resulted. This mating does not appear in the chart. In this kennel no other forms of hernia were ever observed.

A study of the chart shows very clearly that the abnormality was brought into the kennel through outcrossing to carriers. The first outcross was a very famous red sire whose name appears several times in the pedigrees of the other individuals used as outcrosses.

The breed of white bull terriers has been produced by selective breeding for white through many generations. The original stock was probably English, piebald brindle, fighting bull terrier or Staffordshire,[6] with outcrosses to black and tan Manchester and other terriers. The white of the breed represents an extreme development of recessive piebald spotting, so that the only pigment remaining in perfect show specimens is in the eye and on the nose. A careful examination of these white animals will also show small areas of pigmented skin scattered over the body. As a result of this ancestry, even in the most carefully bred strains, many puppies are whelped with some areas of colored coat which are usually located around the eves and on the tips of the ears, and which are known to the fanciers as patch marks.[7] These may be brindle, black, black and tan, or yellow, and dilutions of these. In addition to the patch marks various degrees of ticking crop up continually.

In this breed, as in collies and sealyhams with a similar genetic white, deaf puppies are very common,[8] and this seems to be due to a recessive factor or factors. Many breeders believe that deafness in white dogs is caused by the lack of pigment, but it is just as common in the patch marked specimens as it is

PUPPIES SHOWING HERNIA

Figure 13

Two Cocker puppies showing umbilical hernias. This is the only type of hernia noted in the kennels observed in this study.

in the pure white ones. The lack of pigment theory would seem to be untenable in view of the fact that albino dogs of other breeds have good hearing.[9]

The inheritance of hernia in bull terriers and cockers seems to be similar.

In the bull terrier kennel which we studied only those puppies in which the hernia persisted were noted as herniated in the records. Spontaneous cures therefore would appear on the chart as normals, but any dog obviously blemished or requiring operation before its sale is recorded as herniated, for this reason the chart for hernia is not as accurate as we would desire. Nevertheless it would seem to show that hernia and deafness in bull terriors are inherited as unlinked recessive entities.

Conclusions

1. From the limited data available it is indicated that umbilical hernia is inherited as a specific character independent of other forms of hernia in dogs of several breeds.

2. Umbilical hernia is inherited independently of sex, color, and deafness.

3. Umbilical hernia is a recessive Mendelian character, which, on account of its varying degrees may be due to multiple factors.

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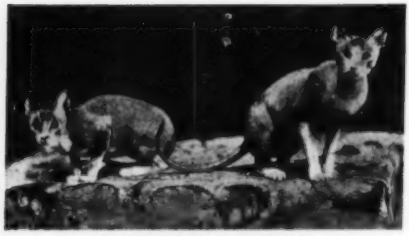
THE ORIGIN OF THE MEXICAN HAIRLESS CAT

THIRTY years ago the New Mexican hairless cat was exhibited at cat shows in this country and accounts of its habits were published by writers of cat books and by people who owned specimens of the breed. Today it is feared that this interesting feline may be extinct. No theory respecting its origin has been heretofore advanced.

The cat commonly was of a maltese color, with a pinkish belly; it was alert, agile, intelligent and affectionate, like other domestic cats, and was said to be fond of a warm bath. Such pictures as we have show the faint stripes on the sides and the beginnings of the letter *M* on the forehead which characterize the ancient type of cat known technically as the striped tabby, commonly as the tiger cat. In size it was either the same as the domestic cat, or about one-fourth smaller. The animal was hairless except that during winter a ridge of fur developed along the back and the upper surface of the tail. It is said to have come from Mexico, though specimens exhibited in the



AN EXTINCT BREED

Figure 14

The New Mexican hairless cat. It undoubtedly evolved from the scant-haired Paraguayan cat, also apparently extinct. From Helen M. Winslow's *Concerning Cats* (1900), by courtesy Lothrop, Lee & Shepard Co.

United States were owned by people living in New Mexico.

The immediate ancestor of this animal undoubtedly was a scant-haired cat of South America, described by the German naturalist, Johann Rudolph Rengger, in his *Natural History of the Mammals of Paraguay*, 1830. Charles Darwin, in *Animals and Plants Under Domestication*, 1868, quoted Rengger regarding the changes effected by climate in the house cat introduced into Paraguay from Europe about 200 years previously; but a misquotation by St. George Mivart in *The Cat*, 1881, caused the animal to be thereafter and down to the present day designated as "the dwarf cat of Paraguay," and prevented naturalists from linking the hairless cat with the scant-haired cat.

Rengger stated that in Asuncion, the capital of Paraguay, the cat had remained much truer to type because of mating with freshly introduced individuals, but that in the interior of the country its habits, coat and size had been affected by climatic conditions to such an extent that the cat sought human habitations only during the rainy seasons, the majority of specimens had become dark gray striped with black, the fur short, scant and close-lying, and "the animal," he said, "is almost a quarter smaller than the cats in Asuncion, though altered males develop to a size equalling that of the European cat."

St. George Mivart made the mistake of saying, "The Paraguay cat is but a fourth the average size of our domestic cat."

Richard Lydekker, in his *New Natural History*, 1896, remarked that the accounts of the cat suggested some affinity with the eyra of the same regions (the eyra being a color phase of the jaguarundi). Our reference books of today therefore incorrectly describe the Paraguayan cat as "not much bigger than a guinea pig, weighing two and a half to three pounds when full grown," and add that "the jaguarundi may be one of its ancestors."

As a matter of fact, the jaguarundi is a natural enemy of the domestic cat and a mating of the two species is said to be structurally impossible.

The average length of the domestic cat, exclusive of tail, is, in the male, twenty-one inches, in the female eighteen inches. The weight of an adult male averages thirteen pounds, of an adult female eight pounds. In a cat "almost a quarter smaller," therefore, the length of the male would be not less than sixteen inches, of the female about thirteen and one-half inches, exclusive of tail. The male's weight would be not less than nine and three-quarter pounds, the female's six pounds. The animals obviously would not be dwarfs. But if those scant-haired cats of Paraguay (now apparently extinct) had been only one-fourth the size of the domestic cat, they would, of course, be no larger or heavier than guinea pigs. Adult cats of this size and weight are unknown.

The size and the almost complete absence of hair in the New Mexican hairless cat both indicate that it descended directly from the scant-haired cat of Paraguay described in 1830 by Rengger. Its faintly discernible markings betray a more remote ancestor in the striped tabby of Asia, which is known to have existed in Europe for at least 400 years; and it was to this type that the majority of the Paraguayan cats had reverted in Rengger's day. He said that the scant-haired cats looked frail. The New Mexican hairless cats also were characterized by a frail appearance. IDA M. MELLEN

COAT COLORS IN HORSES*

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WHILE many excellent contributions have been made to the study of the inheritance of coat colors in horses, there is still some feeling of doubt as to the correct relations between some of the colors. Since so many of the past contributions have been based on studies of herd book data, it was thought that probably additional light could be cast on the subject through other angles of approach.

Method

For a number of years observations were made of coat colors in general of horses, and several hundred individual cases were studied, not with the idea in mind of gathering numbers sufficient to give statistical significance to results, but more as a matter of clarification of certain points in existing theories of coat color inheritance. When a case was observed of a chestnut mare having a bay foal by a black stallion, that phenomenon was established as a fact. Information secured by correspondence and consultations with breeders and herdsmen was used as additional evidence to support deductions and conclusions. Chemical differences were sought by making pigment extractions[5] and attempting synthetic reproductions. Bleaching and colorimetrical tests were made. Cross sections of 53 samples of hair made with Hardy's device[2] were mounted and studied. Photomicrographs of some of these slides are shown in Figures 15 and 16. Much stress was placed on finding physical differences from an histological point of view since this seemed to be a much neglected approach as revealed by the literature.

Classification of Coat Colors

Colors in horses exist in considerable variety and many combinations, often demanding a close study for correct identification. As a rule, however, the colors are readily recognizable. They may be grouped and described as given under the following headings.

A. Basic Colors

I. Black—body color is true black (disregarding weathering):

1. Black—true black without light areas.
2. Seal brown—black with light areas to include muzzle, under eyes, flank, and inside of upper legs, termed "light points."

II. Bay—shades from tan to brown, with black mane and tail and often with black lower legs:

1. Mahogany bay—the brown shades of bay, often called brown.
2. Blood bay—the red shades of bay.
3. Sandy bay—the light shades of bay.

III. Chestnut—shades from yellow gold to dark brown, mane and tail not black but approximately the color of the body:

1. Liver chestnut—the dark shades, some appearing dark brown with an auburn hue.
2. Sorrel—the red shades; sometimes in the lighter shades the mane and tail are of the color described as "crushed strawberry."

IV. Ysabella—a color group having flax or silver manes and tails:

1. Red ysabella—red sorrel-like; flax mane and tail.
2. Palomino—a golden yellow; silver mane and tail.
3. Pseudo-albino—very light cream to white; silver mane and tail; often have "glass" or blue eyes.

Some disagreement exists as to nomenclature of various shades of the basic

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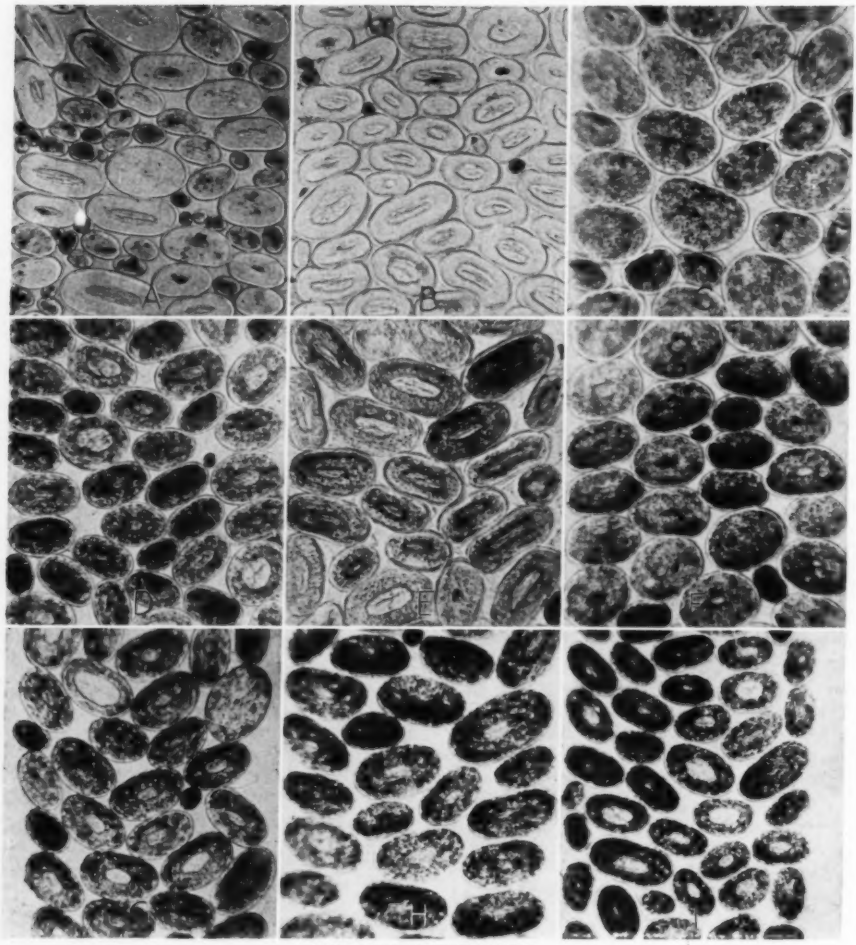
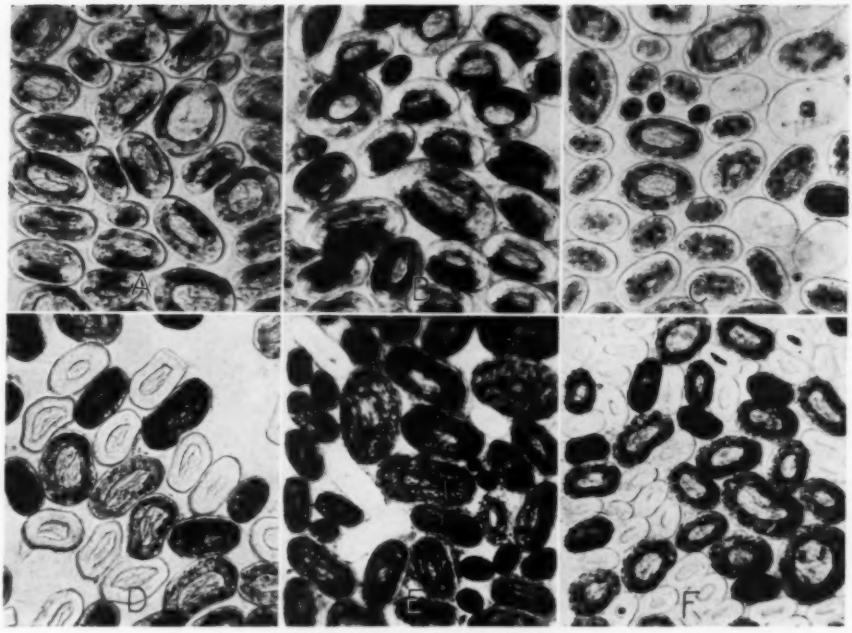


THE BASIC COLORS

Figure 15

There is but one pigment which produces color in hair of horses. The color or depth of color produced depends on the amount of pigment, the extent of clustering of the pigment granules, and the distribution of the pigment granule clusters. The distribution of the pigment granules in the ten basic colors appears to be controlled by three genetic factors. The lightest basic color is pseudo-albino which has traces of pigment; next is the Palomino. *A* shows distal sections and *B* proximal sections of Palomino hair from the same animal. This shows that the pigment in the Palomino horse is concentrated in the tips of the hairs, for in *B* only a few pigmented hairs show, the rest having no pigment in the proximal parts of the hairs. In sorrel or golden chestnut (*C*), the pigment granule clusters are small and evenly distributed. In medium liver chestnut (*D*), there is more pigment and a greater extent of clustering of the pigment granules than is seen in sorrel chestnut. The pigment distribution of sandy bay (*E*), is similar to that of sorrel chestnut, whereas in the darker blood bay (*F*) the arrangement of clusters is about the same as in sorrel, but there is a larger number of them. In light mahogany bay (*G*), there does not appear to be much more pigment than in blood bay, but the clusters of granules are larger. In true black (*H*), there is some variation in the number of pigment granules, but all black hairs have sufficient clustering of pigment granules to prevent light passing through the hair shaft. Seal brown (*I*) is very similar to black in formation of granule clusters; it is essentially black with certain lighter areas.



THE COLOR PATTERNS

Figure 16

The color patterns are made up of hairs differently pigmented, and by different distributions of the pigment in the individual hairs. In buckskin dun (*A*) the crowding of pigment to one side of the hair section is clearly visible. This is characteristic of all hair samples from horses carrying the dun factor. An extreme instance of this is shown in mouse dun (*B*) where the pigment accumulation on one side of the hair is very heavy. In the "light point" (*C*) pattern there is a characteristic absence of pigment in the outer portions of the cortex. (The sample shown is from inside the foreleg of a seal brown mare.) In the roan (*D*), there is a mixture of hairs. (In this sample white, black, and red hairs can easily be distinguished by the different pigmentations. The degree of roaning depends on the proportions of colored and colorless hairs. A distal sample from a blue roan gray (*E*), shows a few white hairs mixed among hairs predominantly black in pigment pattern. Changes in pattern with the shedding of the coat are shown in (*F*), a sample from the same animal as (*E*). This section was made at the proximal end of the hair instead of the distal end and disclosed what appeared to be a white undercoat, as many colorless hairs are to be seen. Since the old coat has been shed, the filly is much lighter, indicating that the new coat exhibited the effect of the gray factor much more than the original juvenile coat did.

colors. The new genetic interpretation formulated by the author is considered in the above listings. Ysabella, commonly considered identical with Palomino, is here suggested as a name for the group of immediately related colors.

B. Color Patterns

I. Gray—causes gradual displacement of colored hair by white hair as age advances:

1. Iron or steel gray—usually a high percentage of colored hair, indicating a young animal.
2. Dapple gray—having the colored hair in such distribution as to give a dappled effect.
3. White—almost devoid of colored hair.

II. At certain ages gray, black roan, and gray roan are practically indistinguishable, but true genetic differences exist. Gray is foaled solid color; any roan is foaled roan; and gray roan whitens with age the same as does gray.

Roan, a more or less uniform mixture of colored and white hairs, occurs in a number of combinations:

1. Black roan—black and white hairs mixed, usually called "blue."
2. Blue roan—usually described as black and white hairs mixed, but almost invariably having some red hairs.
3. Bay or red roan—roaned bay.
4. Chestnut or strawberry roan—roaned chestnut.
5. Paint roan—roaning imposed on the colored areas of paint.
6. Dun roan—roan in combination with the dun factor.
7. Gray roan—roan in combination with the gray factor.

III. Dun—always with dorsal stripe; often zebra stripes on legs and transverse stripe over withers and shoulders; coat appears diluted:

1. Mouse dun—dun imposed on black, seal brown, dark mahogany bay, and dark liver chestnut, giving a smoky effect.
2. Buckskin dun—dun imposed on blood and sandy bay.
3. Claybank dun—dun imposed on sorrel.

IV. Paint or pied—irregular colored and white areas:

1. Piebald—white and black.
2. Skewbald—white and any color other than black.

Gray, roan, dun, and pied or paint may be in any combination.

Discussion of Chemical Studies

In chemical tests it was found that the colorimeter disclosed qualitative differences which were not observed by the unaided eye. These qualitative differences were assumed to be explained by acidification tests which precipitated proteins in the extracted solutions. Here the proportion of pigment in the solution determined the color of the precipitate, indicating that the qualitative differences shown by the colorimeter were due to quantitative differences in amount of pigment. In very small quantitative differences the qualitative differences disclosed by the colorimeter allowed ready comparison. But this difference in amount of pigment in the aqueous extracts did not reach a point where the unaided eye could readily recognize it before the qualitative differences in the colorimeter precluded comparison. These distinct differences indicating quantitative steps and cumulative effects were considered as results of genetic complexes. Results of colorimeter comparisons of extracts are given in Table I.

TABLE I. Colorimeter Comparison of Pigment Extractions

Colors Compared	*Comparability*
Black and Gray (black gray)	+
Black and Seal Brown	+
Black and Black Dun	+
Black and Chestnut	—
Black and Blood Bay	—
Black and Mahogany Bay	—
Chestnut and Black Dun	—
Chestnut and Blood Bay	+
Black Dun and Blood Bay	—
Black Dun and Mahogany Bay	—
Seal Brown and Blood Bay	—
Seal Brown and Mahogany Bay	—
Gray and White Markings	—
Tyrosine + H_2O_2 and White Markings	—
Tyrosine + NaOH and White Markings	+
Tyrosine + H_2O + NaOH and White Markings	+
Stars from Gray and Stocking from Bay	+
All Peroxide-Bleached Samples	+

Of the colors tested colorimetrically, red chestnut and red bay could quantitatively be made to match perfectly without showing even a slight qualitative difference. Of the other colors tested, not one, nor any dilution of a darker color, was found to have qualitative likeness to these two. This is a strong indication that there must be a genetic likeness of some kind in these two colors. Partial explanation will be found in the new hypothesis developed in later paragraphs. The likeness seems to help support a refutation of the hypothesis that bay is black with a restriction factor limiting black to mane, tail, and lower legs.

A likeness is indicated in all colors by the results of the hydrogen peroxide bleaching. This removed the achromatic differences between the colors by lowering the amount of pigmentation in all of them to a diluted sorrel shade which showed qualitative likeness, thus indicating that all colored horses are basically pigmented alike. This is in agreement with the findings in cattle in which Bogart and Ibsen[1] found all to have a gene for red color. Most probably the various colors and shades, except for physical factors, are due to quantitative steps in amount of pigment produced as dictated by the various genes and combinations of genes.

An explanation of the results of the peroxide bleaching is supported by a study of the dopa reaction. (Space does not permit a summarization of the dopa reaction by stages. The reader is referred to Mathews.[3]) This reaction takes place in all horses of color to the extent of Stage 5, which is a red substance and in most horses is carried cryptomerically under the darker effect of completed melanin. It is quite probable that melanogenesis is a continuous process and as a cell keratinizes, this process is halted in the various stages. The amount of melanin completed determines the extent of pigmentation past Stage 5 that the hair will exhibit.

Possibilities in regard to the action of H_2O_2 on melanin and the various stages of incompleted melanin are that the greater part of the whole reaction system may be carried along. The completed melanin is readily oxidized to a colorless substance, the red substance of Stage 5 is either carried to Stage 6 or to melanin and has a similar fate as melanin, and another stage before Stage 5 is carried to the fifth stage to give the red color to the bleached hair. The more probable action, however, is that completed melanin is oxidized into a colorless substance while the Stage 5 red substance for some reason is more stable and is not affected by 24 hours' exposure to H_2O_2.

It is indicated, then, that the amount of melanin resulting (determining the extent of pigmentation beyond that due to Stage 5 red) depends upon the rate of melanogenesis during the life of the cells that constitute the hair shaft. The rate of melanogenesis is most probably dependent upon the action of genes which thus produce various quantitative and cumulative effects.

Discussion of Histological Studies

There is but one pigment that produces color in hair of horses. This pigment is amber color and is deposited in the form of discrete granules more or less fused into irregular shapes and sizes of clusters. No "diffuse" pigment was found. The results indicate that so long as the relative size of the medulla as compared to the cortex is not too great, the presence of pigment or air in the medulla, or the size of the medulla do not affect the color of the hair. Neither does diameter of the whole hair seem to play any important part so long as pigmentation is relative. Color of a given hair seems to be determined by the amount of pigment, the extent of clustering, and the distribution of these clusters in the hair shaft. In these respects the outer portions of the cortex play a particularly important part. The cuticle is evidently too thin and translucent to have an effect in color determination.

In cross sections the fine tips of hairs are noticeably more intensely pigmented than the body of the shaft. This is particularly apparent in Palomino hair (Figures 15*A* and *B*) in which the pigment is very limited in amount. It also becomes apparent in the change of shade when a horse is clipped. It seems that the amount of pigment produced in relation to age in a given hair is the same whether the hair is yet very young and only the true tip of the potential hair, or is at a stage of growth to disclose the largest diameter of the shaft.

Several salient differences in pigmentation between various colors were found. Besides the characteristic mode of pigment production found so noticeable in the Palomino color, it was observed that the variations in color from sorrel to black were accompanied by changes in amount and distribution of pigment as expressed in variations from fairly smoothly shaped, small clusters of pigment granules to decidedly irregularly shaped, large clusters. It is evident that as concentration increases the yellowish or amber pigment becomes more reddish and darker and as the clustering increases the translucency of the hair decreases. This brings about differences in shades. When clustering reaches a certain extent, no light passes through the hair shaft, resulting in black. By the extent of clustering it is possible to distinguish three degrees of pigmentation: the light chestnuts and bays, the liver chestnuts and mahogany bays, and the blacks. It seems possible that the liver

chestnuts and mahogany bays actually do not have any more total pigment per volume of hair shaft than do red bays. The achromatic effect exhibited by the liver chestnuts and mahogany bays is attributed to a higher degree of fusing into clusters of the pigment granules. In the mahogany bays this clustering found expression more in some hairs than in others, resulting in a mixture of black, brown and reddish hairs.

Certain deviations from normal pigment cluster distribution cause differences in color. Hair from all dun horses was found to have the pigment more or less concentrated in one side of the hair shaft, allowing a higher degree of translucency on the other side (see Figure 16*A* and *B*). This gives the coat a characteristic diluted appearance usually recognizable regardless of what the basic color might be. Another type of pigment cluster distribution was found in hairs from light points (Figure 16*C*). These hairs exhibited more or less lack of pigment clusters parietally located in the cortex. Depending upon the degree of expression, this produces color effects varying from dilute brown and tan areas as seen in seal brown horses and some mules to the truly white, mealy points seen in black donkeys (*E. asinus*) and some black mules. Commonly, a roaning effect is produced because some hairs are not affected and so remain darker.

Discussion of Tentative Hypothesis

Planned breeding experiments being out of the question and horse populations in general obviously not being the result of random matings, hypotheses for the mode of inheritance of coat colors in horses must stand on their plausible merit and their ability to withstand refutation.

The evidence indicates that there are three major loci, the genes of which, assisted or hindered by accessory or minor modifying genes, determine the amount of pigment produced. The triple recessive does not produce true albinism but limits the coat color to a very light cream or apparent white and often causes the eyes to be a light blue. These must be the so-called albinos with reduced eye pigment with which Tuff[6] worked, the glass-eyed "albinos" Vaughan[7] gets in his herd of Palominos, and the new breed of horses registered by the American Albino Horse Club.

The presence of one gene for which we use *A* in the first of the three major loci produces the Palomino coat. This is a heterozygous condition as postulated by Tuff, and no doubt explains Vaughan's inability to fix the color of his Palominos in thirty years of breeding. Because of scarcity of pigment in this color, those parts of the hair which are of normal diameter show an amount of color only slightly in excess of white The difference in color of tip and body of the hair, as mentioned, can be recognized macroscopically. This phenomenal pigmentation may be the reason for the manes and tails of these horses appearing practically devoid of pigment. The relation of the Palomino color to dun as suggested by some authors, including Tuff, cannot be substantiated.

The presence of homozygous genes at the first major locus allows a double dose of pigment to be produced, resulting in a red gold, sorrel-like color instead of the golden yellow Palomino. The pigmentation, however, is not sufficient to do more than produce a slightly creamy flax mane and tail. These probably are the "red sorrels" Vaughan has been getting, and probably in combination with the dun factor were Tuff's pale duns. It is possible that genes of the first major locus carry a specific deficiency insofar as mane and tail pigmentation is concerned, which is similar, in an opposite respect, to the ability of a gene at another major locus to produce black mane and tail.

The above three genotypes, i.e., *aa*, *Aa*, and *AA*, are double recessive insofar as the second and third major loci are concerned.

When the first and third major loci are occupied by recessive genes in the homozygous condition, and the second locus is occupied by one or both dominant genes, the result is a sorrel or red chestnut coat color. It is possible that the

difference between the heterozygous and homozygous condition is the difference between the crushed strawberry-maned sorrel and the ordinary red chestnut.

The combinations of dominant genes at the first and second loci determine the shades of dark or liver chestnut. McCann[4] found the sorrel or red chestnut to behave in a recessive manner to the dark chestnut. Naturally, the red chestnuts, not having any dominant gene or genes at the first locus, could not throw any liver chestnut foals, while heterozygous livers could throw both kinds.

When the first and second loci are occupied by recessive genes, a dominant gene at the third produces a light sandy bay. The presence of a dominant gene at this locus is always accompanied by a black mane and tail. The homozygous dominant genes here would still result in a sandy bay, probably a shade darker and with black showing on the lower legs.

When one or both dominant genes of this third locus are accompanied by one or both dominant genes at the second locus the red or blood bays result. When the dominant genes of the third locus are accompanied by the dominant genes at the first locus, the mahogany bays result. In these cases, too, particularly as they pertain to the first locus genes, the shades are the results of the several combinations. It is also probable that accessory or minor modifying genes affect all cases to some extent.

Any combination of dominant genes so long as there is one at each of the three major loci results in black. The presence of only one dominant gene at each locus brings about black, and the addition of other dominant genes or minor modifying genes does not necessarily have any more effect because shades within black cannot be recognized.

A summarization of the genotypes is given in Table II.

The above hypothesis illustrates the mode of inheritance of the basic colors. It precludes blacks and bays from chestnut matings, so well substantiated by all evidence. It permits bay foals from chestnut × black and black × black matings, which are so difficult to explain with the black restriction factor hypothesis.

Except in the lighter end of the color scale, it is very difficult, if at all possible, to recognize the various shades by histological examination of the hair. It is, however, readily possible to distinguish by the extent of the pigment granule clustering between the red chestnuts (Figure 15*C*) and the liver chestnuts (Figure 15*D*), between the sandy bays, blood bays, and mahogany bays, and between all these and black.

Considerable discrepancy exists in the literature concerning the grouping of brown. This study, however, indicates that it should be split into bay and black. The colorimeter tests on the pigment extractions point to likeness of seal brown and black, and much less difference between blood bay and mahogany bay than is shown between mahogany bay and seal brown. Histological examination of cross sections disclosed almost complete likeness between seal brown and black (Figures 15*F* and *G*) and somewhat supported the findings of the colorimeter tests in comparing mahogany bay and blood bay and in showing

TABLE II. Basic Colors and the Genotypes Which Represent Them

Basic Colors	*Genotypes*
Pseudo albino	aabbcc
Palomino	Aabbcc
Red ysabella	AAbbcc
Sorrel	aaBbcc, aaBBcc
Medium liver chestnut	AaBbcc, AaBBcc
Dark liver chestnut	AABbcc, AABBcc
Sandy bay	aabbCc, aabbCC
Blood bay	aaBbCc, aaBbCC, aaBBCc, aaBBCC
Light mahogany bay	AabbCc, AabbCC
Dark mahogany bay	AAbbCc, AAbbCC
Black	AaBbCc, AABbCc, AaBBCc, AaBbCC, AaBBCC, AABbCC, AABBCc, AABBCC

considerable difference between the bays and seal brown. This indicates that mahogany bay should be classed with the bays and seal brown with black.

An explanation of the difference between seal brown and true black lies in a factor which produces light points as found also in mules and asses. The expression of this factor is more apparent in dark colors as a more vivid contrast exists. Histological examination disclosed similarity in hairs from these areas in the two species and their hybrids. This characteristic type of pigment cluster distribution is more pronounced in the ass than in the horse, with the mule being intermediate. This results in the light points of the ass being more nearly white than tan. The mode of inheritance is that of a dominant as evidenced by the literature and by observation. The effects of accessory genes are evident.

The most striking finding in this study was the distribution of pigment in the cortex of dun hair as disclosed by histological examination (Figures 16*A* and *B*). This character is always accompanied by the dorsal stripe associated with dun. It was found on dun horses, basically red chestnuts, blood bay, sandy bay, mahogany bay, and black, and imposed its effect in conjunction with the effect of gray, roan, and pied.

An interesting case observed in this study was that of an apparently roan mare with a buckskin dun mule foal by a black jack. The question of course was the origin of the dun. In spite of her roan color, the mare had a dorsal stripe and when her coat hair was examined under the miscroscope the dun type of pigment distribution was found.

Since every horse found to have this characteristic pigment distribution also had a distinct dorsal stripe there is a good probability that the presence of the dun factor may be recognized macroscopically. Proof can be rendered by microscopic examination.

The mode of inheritance of dun is similar to that of gray, roan, and pied, all of which are dominant to their respective alleles but show no epistasis towards each other. Besides one alone, any combination of two or three, or all four or these factors may find expression in one horse. Degree of expression is more or less determined by the allelic condition and by accessory genes.

Summary and Conclusions

There is but one pigment that produces color in hair of horses. This pigment is amber color and is deposited in the form of discrete granules, more or less fused into clusters of irregular shapes and sizes. The amount of pigment, the extent of clustering of the pigment granules, and the distribution of the clusters determine the color and shade of a hair. Size of medulla, air in the medulla, and pigmentation of the medulla do not seem to have an effect on the color of a hair so long as the medulla does not displace too much of the cortex. Pigmentation in the cortex, particularly in the outer portions of the cortex, is largely responsible for color and shade. The cuticle plays no significant part. Diameter of the hair shaft affects color only if the amount of pigment is not relative. A difference in intensity of pigmentation exists between tips and bodies of hair shafts. This is thought to be due to a constant rate of pigment production during the growth of a hair regardless of its immediate diameter. At least two aberrant types of pigment granule cluster distribution exist. One is associated with a factor producing dun, the other with a factor producing light points.

Coat colors of horses may be divided into two classes: (a) the nine basic colors (p. 437) and (b) the color patterns (page 439). There are other colorations, less common, which were not included in this study. White markings are not considered here.

A tentative hypothesis for the mode of inheritance of the basic colors is proposed. These basic colors are considered to be due to the qualitative and quantitative effects of genes at three major loci. The shades of these colors are affected by accessory or minor modifying genes and gene interactions. Primarily the basic colors are determined by genotypes in the following manner: the pseudo-albinos by the triple recessive, the

Palominos and red ysabellas by *Aa* and *AA*, respectively, at the first major locus only, the red chestnut, including the common sorrels, by the dominant genes, either homozygous or heterozygous, at the second locus only, the sandy bays by homozygous or heterozygous dominant genes at the third locus only, the liver chestnuts by dominant genes at the first and second loci, the blood bays by dominant genes at the second and third loci, the mahogany bays by dominant genes at the first and third loci, and the blacks by any combination of dominant genes at all three loci.

The patterns, gray, roan, dun and light points, are produced by dominant qualitative genes acting independently and showing no epistasis. None, any one, any combination, or all of these pattern genes may find expression on the basic color of one animal. It is possible, and also probable, that there is a difference in effect between the heterozygous and homozygous conditions of these dominant, or partially dominant genes, and also that accessory genes affect the rate of expression of the gray gene, and certainly the degree of expression of the pied, roan, and light-points genes.

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✦

GERMAN GENETIC LITERATURE

BECAUSE of encouragement to the study of genetics and especially to human inheritance given by the present government in furtherance of its policies emphasizing race and race hygiene, heredity has become a very popular subject in Germany.

As a reflection of this increased interest in genetics there have recently appeared in that country a large number of books dealing with this field.

Some of them are popular treatises with a decided eugenic trend. Among these should be mentioned *Die Vererbung* (Heredity) by Günther Just, and *Vererbungslehre, Rassenhygiene und Bevölkerungspolitik* (The science of heredity, race hygiene and population problems) by Hermann Siemens.

Others are compendia of scientific facts concerning human heredity or covering the whole genetic field. Under this group one may place such general efforts as the volumes constituting the *Handbuch der Vererbungswissenschaft* (Handbook of Genetic knowledge). In volume II F 1938 of this series Hans Stubbe reviews general considerations concerning *Genmutation*. *The Handbuch der Erbkrankheiten* is another huge effort. The first volume of this series is on *Der Schwachsinn* (Feeblemindedness) by Dubitscher, and the fifth volume, *Erbleiden des Auges* (Hereditary diseases of the eye), is a coöperative volume jointly prepared by eight ophthalmologists. Then there is the *Studien über Vererbung und Entstehung Geistiger Störungen* (Studies upon the heredity and development of the insanities), volume five of which has been prepared by Friedrich Stumpel to cover the subject of *Erbanlage und Verbrechen* (Heredity and Crime). In this group the late Ludwig Plate's attempt to review the whole field of genetics in the several volumes of his *Vererbungslehre* (Genetics) should also be mentioned.

Specialized fields are covered by the two volumes of Diehl's and Verschuer's *Zwillingstuberkulose* (Tuberculosis in twins). Timoféeff-Ressovsky's *Muta-*

tionsforschung in der Vererbungslehre (Mutation research in Genetics), Max Lange's *Erbbiologie der angeborenen Körperfehler* (Heredeobiology of inborn bodily errors), Wilhelm Weitz' *Die Vererbung Innerer Krankheiten* (The heredity of internal diseases). There are Friedrich Curtius' *Die Erbkrankheiten des Nervensystems* (Hereditary diseases of the nervous system), and *Multiple Sklerose und Erbanlage* (Multiple sclerosis and hereditary predisposition). Reinhold Lotze treats the research methods of twin study in his *Zwillinge* (Twins). Heinrich Kranz gives life histories of criminal twins in *Lebensschicksale krimineller Zwillinge*.

Heredity of the Hand

To this latter specialized type of book belongs Walter Müller's *Angeborene Fehlbidungen der menschlichen Hand* (Inborn developmental errors of the human hand) which is selected here for special comment.

In the foreword Müller directs his one hundred and seventy page monograph to researchers in biology, anatomy and genetics as well as to Rőntgenologists, Surgeons and Orthopedic Specialists. It is of interest that most of the materials have been derived from the author's practice as State Physician for Cripples in East Prussia.

In the first section the author considers variations of the skeletal plan, in which he holds that overdevelopment and underdevelopment of the hand are really different manifestations of the same character which may vary all the way from lack of all digits to an eight or ten fingered condition.

The most common polydactylous form is a doubling of the thumb. This may consist of a rudimentary digit attached to an almost normal thumb or may entail a complete duplication of the whole ray. Duplication of the little finger in a similar fashion is met with somewhat less frequently. The combination of radial and ulnar duplications is rare indeed.

Aside from polydactyly arising from a doubling of individual rays is that produced by a mirror tendency such as is seen in the double-pawed cats so common from Maine to New Jersey.

Müller states that polydactyly is inherited as a dominant, but subject to great changes in degree of dominance. Verschuer suggests multiple allels with different strengths. Ashner and Engleman concluded that there are two linked genes, one for the kind of anomaly and the other for localization. These genes are dominants, and change of dominance can take place within a single family.

Müller recognizes the principle of "space and material." There is a definite tendency for rays weakened by splitting or affected by developmental frailty to fuse with their neighboring rays.

There frequently ensues a fight for "space and material" between two rays that have resulted from the splitting of one original ray, because two rays are demanding development from the materials ordinarily destined to make but one. Frequently one gets the head start, continuing to prosper at the expense of the other.

There may be a degenerative reduction of the ray number. Frequently this consists of a reduction of the thumb ray with a reduction of the metacarpal. Sometimes in these cases the metacarpal fuses with its neighboring wrist bone.

There may be a degenerative defect of the little finger or of the ring and little finger combined. More rare is the degeneration of both little finger and thumb in the same individual. Very common is the reduction of finger number by a fusion of internal rays.

A curious type of defect is the degenerate tibia that brings with it reduction or absence of thumb. A corresponding ulnar degeneration brings a rudimentary little finger. An elbow defect carries with it reduction of the little finger or little finger plus ring finger.

Brachydactyly is frequently combined with a defect of weakened parts. That is to say, the internal rays of digits two, three and four may be involved in an area of general developmental weakness. In this event, fingers two, three and four

differentiate but do not attain normal size.

In Müller's discussion of the heredity of syndactyly, one notes the same error of thought into which many students of human heredity have fallen, namely, a tendency to view all manifestations that are phaenotypically alike as having been produced by the same gene complex. The students of laboratory mammals know the impossibility of distinguishing the homophaenomorphs of the rabbit: "rex$_1$", "rex$_2$", and "rex$_3$"; those of the mouse, "blue" and "leaden" or "wavy$_1$", "wavy$_2$", and "wavy$_3$". For this reason it is surprising to find the author referring to the famous male sex-linked syndactyly pedigree of Schofield as follows:

Mit Recht hebt aber Thompsen hervor, dass mehrfach ganz dieselbe Anomalie auch bei Frauen beschrieben worden ist, und dass er selbst die Anomalie mahrfach bei Frauen ausgeprägt fand, demgemäss muss die Annahme einer Lokalisation des entsprechenden Gens im y-Chromosom doch wohl verworfen werden. (Thompsen shows correctly that frequently the very same anomaly has been described in women by other authors, and that he himself had found it developed many times by women, for which reason must be thrown out the supposition of its determiner being located in the *Y-Chromosome*.)

The second section deals with faulty development on the basis of destruction of the primitive hand. Where there is a destruction of development of the whole primitive hand, smaller fingers may develop. They may all be fused to form a spoon-shaped organ.

A most common form is the split hand in which there is a distal fusion of fingers and loss of some finger bones, but frequently a development of all bones in the hand proper. Hyperplasia or local gigantism of the hands and feet is well known, the tendency to local gigantism sometimes running in families.

The third section deals with inborn destructions to the further development of the hand. Extreme thinness of fingers or arachnodactyly is well known as a hereditary character, as is the general brachydactyly associated with short stature and brachycephaly.

The chondrodystrophic hand form of achondroplastic dwarfs is also described. Exostoses and enchondromatic growths upon the hand bones are figured.

The reading matter is followed by eleven pages of references to the literature.

The book impresses the reviewer as a well balanced treatise of general interest to all, and of great value to those specialists to whom it is addressed.

CLYDE E. KEELER

Howe Laboratory
Harvard Medical School

A LINKAGE BETWEEN SHAKER-2 AND WAVY-2 IN THE HOUSE MOUSE

GEORGE D. SNELL AND LLOYD W. LAW*

Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine

THE following data prove linkage between shaker-2[1.2] and waved-2[3] in the house mouse:†

The crossover percent is 27 in the male, 25 in the female.

	++	sh2 wa2	+wa2	sh2+
†♀ sh2 wa2 sh2 wa2 × ♂ sh2 wa2 ++	25	16	6	9
♂ sh2 wa2 sh2 wa2 × ♀ sh2 wa2 ++	107	85	38	26

1. DOBROVOLSKAIA-ZAVADSKAIA, N. L'irradiation des testicules et l'hérédité chez la souris. *Arch. Biologie* 38:457-501. 1928.

2. CLARK, F. H. Linkage relations of Zavadskaia shaker in the house mouse (Mus musculus). *Proc. Nat. Acad. Sci.* 21:247-251. 1935.

3. KEELER, C. E. A second rexoid coat character in the house mouse. *Jour. Hered.* 26:189-191. 1935.

*Finney-Howell Research Fellow.

ROSCOE B. JACKSON MEMORIAL LABORATORY

Celebrates Tenth Birthday, September 22-24, 1939

THE scene was a sunny room, looking out into the pine forested hills of Mt. Desert Island, Maine. For a longish hour or so there had been a reading of financial and other reports such as sum up a year's activities. Then one of the audience asked for the floor, and this paraphrases the gist of his remarks:

"For forty years I have been engaged in research to discover the causes of cancer. During the last few years in England, cancer workers have been increasingly impressed with the series of papers and reports emanating from the Jackson Memorial Laboratory, whose hospitality I have been enjoying the past few days. So unique and excellent has been the work reported by the Laboratory and so varied have been the problems attacked, that we in England have come to look upon the research being conducted here as among the very hopeful approaches to the basic problem of the causes of cancer. Considering these facts, we imagined that the institute from which this material came must be large, imposingly housed and elaborately staffed.

"You can appreciate my amazement, therefore, to find it so modestly and simply housed and so informally conducted. All the essentials are here, as testified by the high quality of the work produced. But there are no expensive extras, and the fine cooperative spirit of the staff I have found altogether charming and most stimulating during my visit here.

"Certainly on this tenth birthday anniversary, it is a pleasure to testify to the high regard which we hold the work of this Laboratory in England and to my own pleasure at seeing at first hand the unique and most effective establishment you have built up here in the past ten years."

The speaker was Dr. William Cramer, Head of the Imperial Cancer Laboratories in London. His spontaneous testimony to the value of the work done during the ten years that the Laboratory has been in existence, brought into clear focus the contributions which have been made by this unique institution during the troubled decade since it opened its doors. Dr. Cramer's unscheduled speech marked the clumination of a most interesting three-day "birthday party" tendered by the Laboratory to some of its friends in the fields of mammalian genetics, and of cancer research.

Excerpts from the report of the Laboratory, which will be given below, give the present status of research projects in progress. A few historical notes may not be amiss.

Beginnings

The Laboratory is the culmination of a dream of its Director, Dr. C. C. Little, to attack the problem of the genetic basis of cancer through the production of uniform strains of mice differing in cancer susceptibility. During the late 1920's Dr. Little interested Mr. Roscoe B. Jackson, President of the Hudson Motor Company, in the project. Plans were prepared for a building to house the laboratory, and for the support of its program, when the sudden death of Mr. Jackson hit like a bolt of lightning. The blow was parried by Mr. Jackson's family and his friends, who financed the building of the original Laboratory, and by other interested doners who contributed funds to start it in operation. In 1929 the building was completed and the Laboratory opened its doors.

The purpose of the Laboratory was to attack some of the basic problems of cancer genetics which had been only partly elucidated through genetic experiments up to that time. One of the great needs in coming to grips with the genetic basis of cancer, was the production of a number of pure lines of mice differing in their cancer susceptibility. So at the start the Laboratory became a center of mouse inbreeding. Basically this has been of tremendous importance in cancer research because it has made available standardized genetic units in a species which suffers from practically all of the kinds of cancer human beings are heir to, and cancers so similar to human cancers that they represent from the histologist's point of view practically exact parallels. Since the mouse has a life span of only three years, coming into the "cancer-age" in from ten to sixteen months, it is obviously ideal experimental material to attack not only pure genetic problems, but effects of diet, carcinogenic agents, etc. In all of these other research areas genetically uniform mice were essential, for inequivocal answers to the questioning of experiment were virtually unobtainable as long as the genetic background remained an unknown variable.

So, just before the depression began the Jackson Laboratory set up shop by assembling strains of the most highly inbred mice then available, and by making plans to continue them and to develop still more pure lines of mice. At the same time, it was proposed to push forward on genetic problems as rapidly as possible with the material then at hand. A number of very significant research projects have gone forward to most important conclusions. Their present status is given in the excerpts from the Report quoted below.

The Depression

The original sponsor of the Laboratory, in whose memory it is named, as before mentioned, died before its completion. Then came the depression: and the contributions necessary to meet the overhead of the Laboratory practically disappeared. Faced with a major emergency, the staff of the Laboratory courageously decided to go on with their work, if not entirely without funds, certainly without enough funds for even basic needs. So serious was the situation during 1931 and 1932, that the staff was put on a part-time basis, the rest of the day being devoted to gardening, fishing and work at home tc produce and preserve the food necessary for the needs of staff members during the summer and winter, and to save personal expenses. The Laboratory was kept going and stocks were kept intact under most discouraging conditions.

Since that time financing of the Laboratory has been a serious problem, for it has no endowment. It has one important asset which has been put to good use to furnish cash to carry on the work, and at the same time to speed the progress of genetic research. The inbred mice reared at the Laboratory are unquestionably the most uniform mammalian material available today. Conceivably a single strain of the Wistar rats or of the Department of Agriculture's Guinea pigs is just as uniform as any inbred mice at Jackson Laboratory. But *the variety of uniformity* to be found there is unique. There are available some twenty-five strains which have been inbred 12 generations or longer. The *A* strain has been inbred for over fifty generations. Thus for laboratory and for genetic experimentation a large number of different genotypes are available in quantity,—uniform material of known behavior with respect to cancer.

This asset was used to meet the acute financial emergency the Laboratory went through during the depression. Through the sale of pedigreed mice of known genetic constitution a cash income has been obtained when no other funds were to be had. From a small beginning this unique mouse business has grown to the point where last year the sale of mice

netted enough to take care of about half of the overhead of the Laboratory. Genetically this traffic in mice has considerable importance too, because the greater the number of mice reared, the better are the chances of finding mutations,—and mutations are essential to genetic research, such as linkage, etc. The large number of mutations discovered in these inbred mice at the Jackson Laboratory is a direct result of quantity production on a scale impossible for purely experimental purposes.

Value of Uniform Strains

Such a central source of uniform genetic material has another very important angle. No inbred strain stays the same, generation after generation. Mutations occur and even the most highly inbred strains are probably not homozygous for all genes. Thus there must inevitably be changes even in this most uniform of mammalian material. The Laboratory makes continuous efforts to maintain the genetic homozygosity of the strains, as far as possible by observation, bio-assays based on transplantation of tumors, etc. But in practice, such inevitable variability of inbred strains is not as serious as it might be, if all research in a given problem is carried on with inbred strains of mice bred in the same laboratory. By such standardization much of the present unknown variability will be eliminated. The importance of this fact is just beginning to be realized by research workers in other fields such as nutrition, immunology, etc. As this basic need for standarized material is more generally accepted, the value of the Laboratory to science will increase, as will the support of the Laboratory through the sale of mice. Certainly in a world which is becoming increasingly unsettled and uncertain, a research laboratory which is able not only to do a vitally important pioneer job of biological standardization but at the same time is able to assure itself a cash income through such service is very fortunately situated.

It is significant that the most important development to come out of the Laboratory to date is the discovery which has been made regarding the transmission of breast cancer. These experiments have proven that in mice some carcinogenic element specific for breast cancer is carried in the mother's milk. This opens up the distinct possibility that certain types of human cancer might be transmitted in the same way. It follows that by fostering or by artificial feeding the transmission of such cancers could be stopped in a generation. It is foolish to attempt to argue direct from mice to men, but here is one lead which certainly should be considered in dealing with human cancer and which might have very important implications. Very significantly, the breast cancer problem in mice turns out not to be exclusively genetics, but also a matter of "internal environment." This emphasizes again, that it is impossible to predict in advance the value of basic research, which gives so often unexpected leads and unexpected applications in quarters which that could not possibly have been anticipated from previous experience.

It is hardly necessary to add to Dr. Cramer's testimony as to the value of the work done. The following excerpts from the Tenth Anniversary Report shows how matters stand at the moment with research now in progress.

Excerpts from the Report of the Roscoe B. Jackson Laboratory for 1938-39

Dr. John J. Bittner has consolidated and extended the evidence demonstrating the influence of the milk of foster mothers on the incidence of breast cancer. His findings have been confirmed in several other laboratories and form one of the most interesting and important discoveries recently announced in cancer research. An arrangement is being completed by which the chemistry of the milk can be investigated in cooperation with the Department of Chemistry of Yale University. Combining the evidence obtained from the foster nursing experiments with data of other types, Dr. Bittner has recently advanced a theory to cover the cause and origin of breast cancer in mice. Three elements are involved in this theory.

1. The milk influence to which reference has been made.
2. A single dominant Mendelian factor.
3. Influence of glands of internal secretion, especially the ovary.

This theory is capable of an experimental test and is now under further investigation.

Dr. Bittner has also obtained much more extensive data on lung cancer and has found that figures which he obtains conform with the theory that the susceptibility to lung cancer in mice depends upon a single dominant Mendelian factor. The completeness of dominance and the extension of the theory to other strains and more advanced generations is now under investigation.

Dr. Arthur M. Cloudman has continued his studies in connection with the extremely interesting liver tumor mentioned last year, and has obtained further evidence of the possible existance of some extra-cellular influence which is operating. He has also spent a great deal of time in the diagnosis of tumors obtained in various experiments and has supplemented in an extremely valuable way the genetic work being done by other investigators.

He is carrying on a considerable number of useful and important transplantable tumors which are and will be used in various investigations. He has acted as Associate Editor of the Bulletin of the American Society for the Control of Cancer and as Treasurer of the Laboratory. In both positions he has shown real ability and has accomplished valuable results.

Miss Elizabeth Fekete has completed for publication her work on the microscopic conditions characterizing the earliest stages of breast cancer and also her work on extended and prolonged nursing in relation to the incidence of breast cancer. She has continued her experiments with transferring fertilized ova in mice and has begun an interesting and important series of experiments on the effect of feeding young mice with ovarian secretion.

Dr. Walter Heston, who is beginning his second year as a United States Government Research Fellow, has collected a considerable amount of data on the inheritance of susceptibility to lung tumors in mice. He is hunting for evidences of linkage between the genetic basis for these tumors and other known genetic factors. By using chemical agents which influence the formation of cancer he has been able to hasten its appearance and so obtain much more complete data in a shorter period of time.

Dr. Lloyd W. Law, as a Finney-Howell Foundation Research Fellow, has been carrying on experiments on the injection of carcinogenic chemicals into embryos before birth and into very young mice. A lapse of further time will be necessary in order for final results to be obtained.

Dr. William Russell has studied intensively the various morphological abnormalities occurring in the C57 black strain of mice and has analyzed them along the lines outlined by Dr. Sewall Wright in his extensive studies of somewhat similar types of abnormalities in other rodents. He has also made certain studies on pigment changes following the feeding of mice on a diet completely free from copper. These latter experiments are in a preliminary stage, but have given interesting results. He has also been building up a large number of genetic stocks for further study of the metabolism of pigment formation, an important matter both to genetics and to general physiology.

Dr. Elizabeth Russell, who has obtained a fellowship from the Association of University Women, has done excellent work on the several tumors found in the larvae of the Fruit Fly Drosophila. She has developed marked skill in transplanting these tumors from one larva to another and has obtained evidence concerning the nature of the embryological changes which indicate that a tumor is about to be formed.

Dr. George D. Snell has completed the tabulation of the effects of sterilized day old mice by X-rays and has found that this process markedly influences the amount and type of tumors formed by these animals in later life. He has carried on a number of extensive studies on the embryology of mice and has obtained information which clears up a number of doubtful points in that process. The studies have been made more valuable by the preparation of photographic illustrations which will be of lasting value. He has also studied the process of ovulation in mice and by regulating the amount of light and darkness in which the mouse lives, found an interesting relationship between that factor and the process of liberating the eggs from the ovaries. He is also making plans for a lengthy study of the effects of diet on the production of mutations in both mice and in the fruit fly.

Dr. George W. Woolley has collected sufficient data to enable him to obtain interesting information concerning the extreme dilution strain of mice in which a high incidence of ovarian and testicular tumors has been found. These mice also show interesting abnormalities of a physiological nature affecting their real productive capacity which may possibly be correlated with the tumor forming process as observed in them. He has also studied further the effects of the male sex hormones upon the incidence of breast cancer both by the production of a large series of parabiotic mice and by the implantation of testicular tissue in the ears of similar mice. Final data on these experiments await the death of the animals now under observation. He has obtained important evidence of adrenal changes in ovariectomized mice associated with breast tumor formation.

The Director has completed a preliminary study of the effects of a species hybird cross

on the incidence of tumors. Using the Asiatic mouse, *Mus bactrianus* and the C57 strain of ordinary mice, *Mus musculus*, a first hybrid generation was produced. The hybrids show a very marked increase in the incidence of various types of tumors other than those of the breast and also a much greater tendency to form multiple tumors. This indicates that widely divergent physiological types of mice produce hybrids which in their old age show upsets in the growth process leading to the formation of tumors more frequently than in the case where the parents resemble one another physiologically. He has also completed for publication a manuscript on the genetics of the various types of tumors other than those of epithelial origin. He is at present conducting an extensive series of experiments repeating the cross between high and low breast tumor strains of mice with a view to following these and studying them more in detail in generations subsequent to the first and second hybrid generations. He was appointed Chairman of the section on growth of the International Genetics Congress at Edinburgh which he was unfortunately unable to attend, although he arranged the program of that section. He was also appointed the chairman of the section on genetics at the Third International Cancer Congress at Atlantic City this month. He has been elected President of the American Association for Cancer Research for a second time, to serve in 1940. His first term was in 1930.

Excellent progress has been made in preparing and assembling the material for the book which was mentioned last year as being supported by a grant from the Markle Foundation. It is expected that the book will be ready for publication next spring. A good balance from the appropriated funds remains to assist its publication. (This book parallels for the mouse Donaldson's unique work on the rat.)

* * * *

We should not, however, be blind to the fact that the most serious financial problem still remains before us. This is the stabilization of its support and the eventual removal of that factor from the realm of uncertainty to that of reliable income. It is needless to emphasize the fact that the present European war has introduced a variable entirely out of our control which must be reckoned with. It seems that that factor has once and for all eliminated from present consideration the possibility of one or a few large donations to solve the problem of permanent financing. With this fact in mind the Director is constantly engaged in planning and organizing efforts to contact as many sources of financial support as possible. These naturally include the possibility of relations with existing education and research institutions, the obtaining of grants-in-aid or large donations wherever possible and the building up of various methods by which larger numbers of small donors can be reached. All of these things require more energy and ability than any one man can possess. For this reason the Director urges every member and associate of the laboratory to help in the progress of these efforts. No matter how small or insignificant a source of support may appear, it must not be neglected. We should not forget that larger sums can be accumulated by building up an extensive list of small donors and that few people realize that the laboratory is not endowed or adequately supported.

The Director does not intend to neglect already existing sources of support and wishes especially to emphasize the value of the sale of surplus mice as a back log in the financial receipts of the laboratory. There is no reason why this program should not be made to pay at least one-half of the laboratory's needed budget. Roughly speaking, it would seem that after the work of supplying mice to other institutions has been efficiently organized, a balance of some $25,000 will remain to be found from other sources. While this seems like a large sum of money when considered as a unit, it does not take many grants or donations of $1,000 to $3,000 before the sum can be reached. It is hoped that the present donors to the laboratory will continue or extend their gifts. If this happens our problem of finances will be reduced to approximately $10,000 per year, which is a distinctly different matter from raising the larger sum.

—Robert Cook.

◆

An Italian Genetic Text

THE list of up-to-date and comprehensive text-books on genetics is increased by one* from an instructor at the University of Rome. The general treatment of the subject is "orthodox" and shows a balance and a critical faculty which were notably lacking in some of the older Italian textbooks on this subject. Application of genetics, as in agriculture and eugenics, are dismissed with little more than a reference, but adequate space is given to some of the philosophical and evolutionary aspects. Good bibliographies and indices also represent an advance over the very sketchy methods that have been used in the past by some Italian authors.

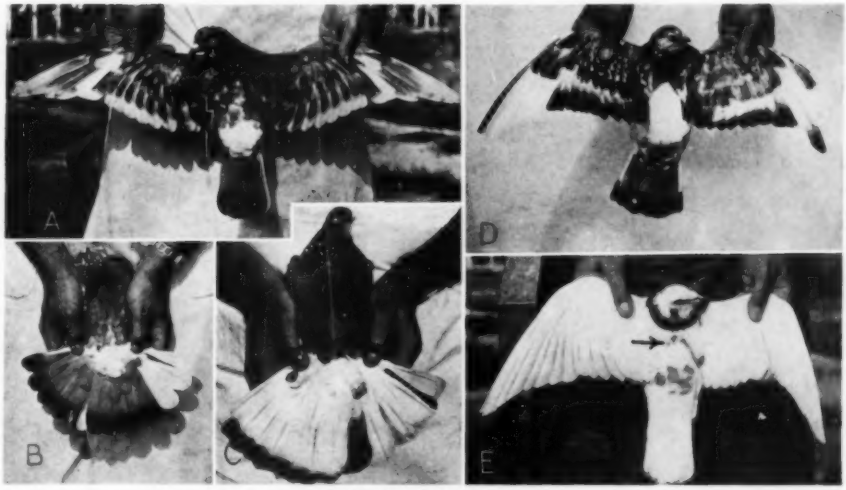
Paul Popenoe.

*Montalenti, Guiseppe. "Elementi di Genetica." Pp. 453, with 122 illus. Licinie Cappelli, Bologna, Italy.

STRUCTURAL ANOMALIES AND COLOR MOSAICS

Observed in a Colony of Domestic Pigeons

WENDELL M. LEVI AND WILLARD F. HOLLANDER



MOSAIC PIGEONS

Figure 17

A-C—a blue-tail mosaic appearing in a male racing Homer hatched in 1938. The juvenile plumage (*A, B*) was ash-red, except most of the tail and the back of the neck, which were typical blue. In the adult plumage (*C*), the pattern was the same except that only one tail feather was blue. The change in color between the juvenile and adult plumage suggests that some hormonal factor also is involved. *D* is a blue-grizzle mosaic, in a crossbred female (white Carneau male × blue-Homer female). The blue checkered left wing is a pattern not normally found in this cross. *E* is a slightly piebald Homer. The colored feathers are blue except for about three ash-gray feathers on the back, suggesting somatic mutation.

IN almost any large population of animals, freaks may be observed, and information about them is always of interest and scientific value. At the Palmetto Pigeon Plant of Sumter, S. C., scores of thousands of pigeons of several kinds are examined every year. We have taken advantage of the opportunity and present herewith case histories and portraits of some of the most unusual specimens which have occurred in the last few years.

Feather Peculiarities

1. "PORCUPINE FEATHERS" (Figure 19*A*) developed on a Female Homer hatched in 1931. Plumage condition is similar to a recessive trait studied by Cole and Hawkins.[2] Color red and ashy.

Pedigree: Parents normal, brother and sister; of Logan strain, with pedigrees back to 1920; (all) ancestry racing Homers of World War stock.

Progeny: At Palmetto, with normal Homer male, produced a large number of young, all normal. At University of Wisconsin, with true "porcupine" male, produced five young, all normal. Grandchildren from out-crosses of sons all normal, numerous.

Comment: The trait may be heritable, but, as no affected young were produced with the previously known "porcupine," the two can not be identical. The bird died at the age of six years. She had a tendency to lay double-yolked eggs, and died with such an egg in the oviduct.

2. "SILKY" (Figure 19*B*) was ob-

WING, TAIL, AND EYE DEFECTS

Figure 18

A-C show dorsal, ventral, and x-ray views of the "wingless" variation. The humerus bones are normal; the rest of the wing being rudimentary. *D* is a "Rumpless" White Carneau female. *E* shows a rumpless and a normal squab at an age of about 2½ weeks. *F* is a squab with abnormal eye lids, about 7 weeks old.

served in a male crossbred, hatched in 1938. Color pure white. Plumage condition apparently the same as silky known for centuries in fancy pigeons.[5]

Pedigree: Father, White Carneau; mother, "blue" racing Homer. Silkiness unknown in parental breeds.

Progeny: With an unrelated racing Homer, four silky and two normal young; with a White Carneau of separate family but of same strain, one silky and three normal young.

FEATHER AND BONE DEFECTS

Figure 19

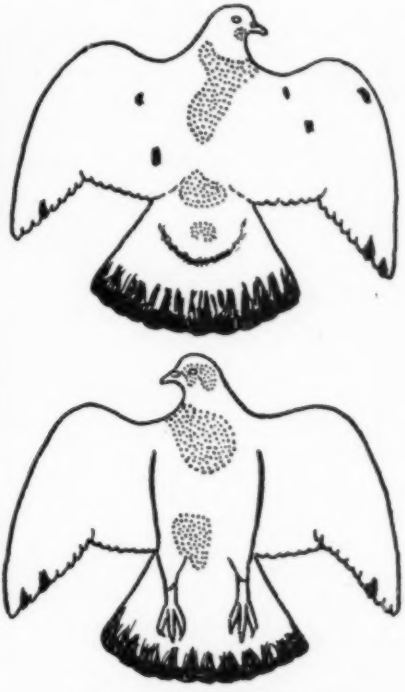
A—"Porcupine"-like female Homing pigeon. A cross with the previously known Porcupine gave normal young. This proves that if this variation is inherited it is different from the previous "Porcupine." *B*—A silky feathered male crossbred. Equal numbers of silky and normal offspring were obtained through mating this individual with normal. *C* shows breast bones of "split-keel" and as compared with the normal bone formation (*D*). The bird having this defect is shown at *E*, with the breast feathers removed.

Silkies both male and female. Total: five silkies, five normals.

Comment: The trait is evidently a simple dominant mutation; it may be identical genetically with previously known silkiness,[8] also a simple dominant trait.

Skeletal Anomalies

3. "SPLIT-KEEL" (Figures 19*C* and *D*) was noticed in a female White Carneau, hatched in 1936. Halves of sternum are united by cartilage only, at anterior end; practically no keel; viscera unprotected on ventral side; heartbeat observable; flight poor.



A BLUE-BROWN MOSAIC

Figure 20

Dorsal and ventral diagrams of juvenile plumage of a male King pigeon. Apparently the mosaic was due to a loss of the "blue" allele in the regions showing some brown plumage (stippled).

Pedigree: Parents normal.

Progeny: At University of Wisconsin, with common pigeon, produced eight young, all normal, and of both sexes. Son × mother gave four young, all normal. Son × daughter gave three young, all normal.

Comment: No evidence of heritability so far. The trait has appeared occasionally in the population, however, about six cases. One similar cock specimen failed to breed because of inability to copulate.

4. "WINGLESS" (Figure 18*A-C*) was observed in a Female White Carneau, hatched in 1936. Wings are rudimentary—humerus and small bones only. Tail has eight instead of twelve main feathers; no oil gland, three instead of seven tail vertebrae (by X-ray study). Left foot slightly clubbed inner and rear toes closely joined.

Pedigree: Parent normal.

Progeny: Most eggs infertile; one hatched

and seemed normal but died early.

Comment: This bird lays only one egg to a clutch instead of two. Several squabs with one rudimentary wing have previously appeared in the population.

5. "RUMPLESS" (Figure 18) affected a Female White Carneau, hatched in 1936; no tail vertebrae or oil gland. Feathers of this region similar to normal tail coverts.

Pedigree: Parent normal.

Comment: No similar pigeon has previously been described to our knowledge. Breeding tests are just being started.

Abnormal Eyelids and Eyes

6. White Carneau squab, hatched in 1938 (Figure 18*D*). Eyelids extensive and immovable; fringed edges lacking. Nictating membrane functional, eyes normal.

Pedigree: Parents normal.

Comment: The squab was weak and lived only to the age of two months; sex not determined.

Color Mosaics

7. "BLUE-GRIZZLE MOSAIC (Figure 17*A-C*) was observed on a female cross-bred, hatched in 1937. Plumage grizzled (*G*-factor[1]) except for blue checkered left wing; moderately piebald.

Pedigree: Father, blue-checker racing Homer, no white feathers; mother, grizzled ash-red and piebald, cross-bred from White Carneau male × blue Homer female.

Comment: Apparently the grizzle factor was eliminated in the left wing during development.

8. "BLUE-RED MOSAIC (BELLNECK)" (Figure 17*E*) characterized a male racing Homer, hatched in 1937. Plumage highly piebald; colored feathers "blue" except for about three red-ashy feathers on back.

Pedigree: Both parents ash-red (B^A, sex-linked factor, Hawkins.[4]) Mother piebald in addition.

Progeny: With a "blue" Homer (recessive to B^A), five squabs: three ash-red and two "blue."

Comment: Even though this mosaic was at first mistaken for a "blue," he breeds as heterozygous for the dominant B^A factor, as would be expected from his parentage. Apparently, the B^A factor was eliminated from most of the skin during development.

9. "RED BLUE-TAIL MOSAIC" (Figure 17*D*) was noticed in a male racing Homer, hatched in 1938. Juvenal plumage ash-red (B^A) except that most of tail and back of neck is typical "blue"; slightly piebald. Adult plumage — same except only one tail feather now "blue."

Pedigree: Father, blue checker racing Homer; mother, ash-red and piebald racing Homer.

Comment: The expected plumage color of this bird is ash-red. The difference between juvenal and adult plumage indicates that something in addition to genetic loss or alteration is responsible for the appearance of the "blue" color.

10. "BLUE-BROWN MOSAIC" (Figure 20) was observed on a male King, hatched in 1938. Piebald with large patches of brown plumage on head and body; "blue" spots in wings; juvenal tail plumage "blue," but white in adult.

Pedigree: Mother, White King; father, "blue" King, son of the same White King × "Silver" King. "Silver" in the King breed is the effect of the sex-linked recessive[4] brown or chocolate factor *b*. The White King used is genetically "blue."

Comment: Apparently the mosaic had the same genetic constitution as its father except for the piebaldness, but the *b* factor was exposed in parts of the skin by loss of the "blue" allelomorph.

Summary

Six pigeons are described which show structural anomalies of feathers, skeleton, or eyes. Of these, one is a male, four are females, and one of unknown sex. The male, having silky plumage, is shown to have a dominant mutation; inheritance has not been demonstrated in the other cases.

Four color mosaics are described; in each case, known color factors are involved. Each mosaic appears to be the result of loss of the dominant allelomorph in part of a heterozygote. It is interesting to note that each mosaic shows more or less piebaldness, and that the piebaldness is always traced to the mother. The significance, if any, of this fact is unknown; piebaldness is due to factors more or less dominant but as yet not thoroughly analyzed.

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European Journals and the War

THE non-receipt by a subscriber of any European chemical or other scientific journal seriously needed as research material should be promptly reported to the American Documentation Institute.

The Cultural Relations Committee of ADI, which cooperates closely with the Cultural Relations Division of the Department of State, is working on this problem, and hopes to be able to surmount such war obstacles as interrupted transportation, embargoes and censorship, which so grievously affected the progress of research during the last war.

The principle should be established, if possible, that the materials of research having no relation to war shall continue to pass freely, regardless of the countries of origin or destination.

Reports, with full details of where subscription was placed and name and address of subscriber, volume, date and number of last issue received, should be addressed to: American Documentation Institute, Bibliofilm Service, care of U. S. Department of Agriculture Library, Washington, D. C.

One "Newspaper Hybrid" Pays Off

ONE of the minor trials and tribulations of the editor is to keep track of and mostly to discount and "debunk" the amazing hybrids which fertile and unhampered imaginations manage to invent, and attempt to perpetuate through medium of publication in the daily press. Sometimes the basic ingredient of the hybrid is simply ignorance, as witness the "corn-lima bean hybrid" widely featured in illustrated sections of the newspapers a few years ago. This was nothing more than an ear of corn badly infested with smut,—an object well known enough, and common enough to suggest that the photograph must have been sold to the newspapers by a practical-joking "hick" taking the "city slickers" for a ride. And how some of them did *ride!*

When the Editor recently was regaled with clippings describing a "moose-cow hybrid produced by the University of Minnesota," he pricked up his ears in real earnest. Such a wide cross, produced by one of our great experiment stations would be an interesting bit of research indeed. Having suffered much serious disillusionment before, he embarked on his inquiries with faint-hearted cynicism, expecting to find rather less than nothing. He was therefore much surprised to be informed by Professor Winters that while the university had not "produced a moose-cow hybrid," it did have under observation a curious creature alleged to have this unconventional ancestry. So close an approximation to a verifiable fact in connection with a "hybrid-yarn" was unusual enough of itself to be "news." While Dr. Winter's two colleagues are very doubtful that a bull moose is to be found in the pedigree of the "moo-cow," the story they have to tell about this animal is interesting in itself, and it may lead to discoveries of definite value regarding genes or endocrine glands (see page 458).

While we have to be cautious in accepting popular claims of unconventional hybrid animals this most interesting contribution does show that frequently at the end of the hybrid rainbow is to be found, if not a pot of genetic gold, at least a handful of the currency of the genes which may be a valuable and useful addition to knowledge. The authors note that two animals having a similar syndrome of malformations have been reported. Very likely the popular conviction that all queer looking animals are hybrids will again in this case foster the preservation of valuable scientific material.—EDITOR.

A MOOSE-DAIRY COW CROSS---?*

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A REPUTED natural cross† between a wild moose and a dairy cow was secured from a dairyman situated a few miles north of Duluth, Minnesota. Externally the animal presented some characteristics which indicated mixed parentage although most of the phenotype suggested dairy breeding (Figure 22).

The head (Figure 22*C*) presented only one abnormality in that the animal was blind in both eyes. Upon autopsy it was found that the lens portion of the eyes was either deficiently developed or absent. Both organs were saved for histological study. The head bones, horns, and tooth pattern did not deviate from a normal dairy animal.

The vertebral column of the animal presented the only skeletal abnormalities found. These were also observable in the live animal (Figure 22*A*). The dorsal processes of the thoracic vertebrae were slightly shortened and a definite elevation was noted at the junction of the thoracic and lumbar regions. Both the loin and sacral regions were curved dorsally, and the sacral vertebrae were distinctly curved ventrally. As Figure 21*A* indicates, the body portion of some of the lumbar vertebrae were ventrally wedge shaped in contrast to the normal parallel features of the articular surfaces. The vertebral foramen of the lumbar and sacral vertebrae were unusually flattened dorso-ventrally.

The first three segments of the sacral vertebrae were not fused. However, the fourth and fifth segments were fused and the units abbreviated. The dextrolateral border of the fourth vertebra was curved ventrally and the same border of the fifth segment was discontinued. The first coccygeal vertebra articulated dextrolaterally to the last sacral unit, while the second coccygeal bone articulated with the last sacral vertebra and the first coccygeal unit. The second and remaining coccygeal vertebrae assumed a 45° sinistral angle in respect to the sacral bones (Figure 21*B*). The second and third coccygeal vertebrae were fused as were some of the other units.

Posteriorly, the anus assumed a dorsal position as Figure 22*B* indicates. The scrotum was posteriorly shortened so the apex assumed a posterior rather than ventral position. Two well developed (for the male) teats were located on the anterior surface of the scrotum. No teats were present on the abdominal wall.

Both testes were well developed for a dairy animal of this age (approximately one year). In the moose, the testes are small during the summer. Therefore, because of the slight excess size of the organs even when compared to dairy bulls, it appeared that the testes and associated endocrine functions did not resemble the moose in any respect. Sections of the testes were fixed for future studies on possible meiotic or chromosomal deviations.

The pelvic girdle was small compared to that of dairy bulls of the same size and age, as Table I indicates.

In addition the tuber ischii was prominent. The narrowness of the posterior

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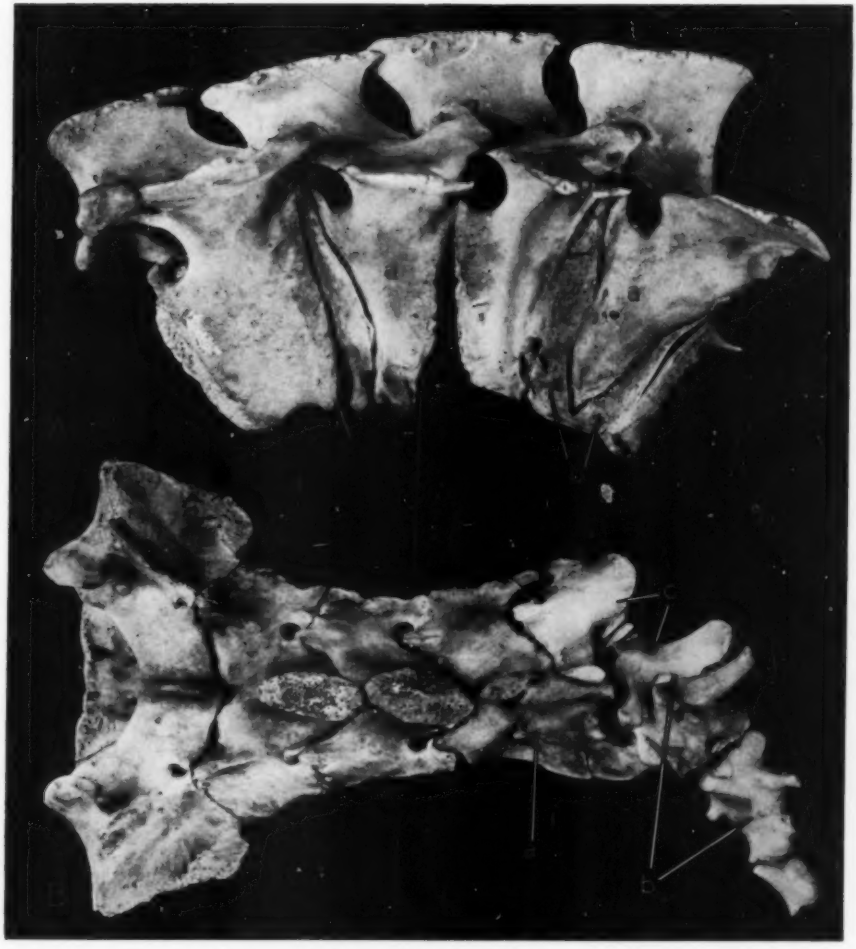
†The animal was furnished through the courtesy of Mr. T. H. Fenske, Superintendent of the West Central School and Experiment Station of the University of Minnesota, Morris, Minn.



VERTEBRAL DEFECTS

Figure 21

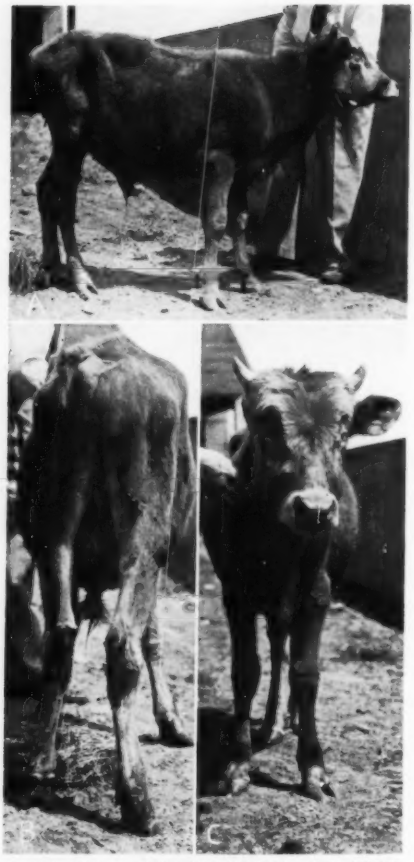
A—Lumbar vertebrae, showing the wedge shape of the body portions (a) as well as the fusion of some of the vertebral discs. *B*—sacral and coccygeal vertebrae, showing the fusion of the last two sacral units (*a*). The malformation of the coccygeal vertebrae are evident in the fusion of some portions (*b*) and the displacement of the first and second bones (*c*).

pelvic bones resembled the moose slightly more than the dairy type.

The autopsy was conducted by one of the authors (R. F.) who has had considerable experience with both moose and dairy types. All internal organs were typical of the domestic species. The spleen was large and elongated, as is usually found in dairy animals, in contrast to the moose spleen which appears more oval and comparatively small in size.

The hair evidently had a chemical composition similar to the dairy animal as one of the workers (R. F.), who is very sensitive to moose protein, experienced no distress while working with the animal.

The meat was of a light color and did not exhibit the dark color of moose meat



EXTERNAL APPEARANCE OF THE REPUTED GENUS CROSS

Figure 22

A—Side view. The vertebral deformities are discernible. *B*—Rear view. The abbreviated tail, dorsal anus, and shortened scrotum may be noted. *C*—Front view. The usual dairy horns are present. The opacity of the eyes may be seen in the right organ.

even after storage. The cooked meat had the typical flavor of young beef.

The animal was skeletonized and all osseous components were similar to those of the dairy animal.

A purebred Shorthorn female very similar to the one here described was seen recently by Prof. W. H. Peters of the Animal and Poultry Husbandry Division of the University of Minnesota. The female presented the usual Shorthorn characteristics as to color markings and head features. This animal had the same type of depression of the top line between the shoulders and loin. The loin and sacral regions were arched the same as the autopsied animal. There was no tail. The vertebral column ended with the last sacral vertebra. The anus was placed dorsally with the vulva immediately posterior to it. The eyes were normal.

In the autopsied animal, all the features studied, with the possible exception of the width of the pin bones, were similar to the usual dairy conformation. The conditions found could have been the result of a species cross, embryological malformation, or a genetic deviation, most likely the latter. Figure 22*C* shows a set of well developed dairy type horns. Structurally these did not deviate from the usual *Bos taurus* horns. The interesting phenomena of the presence of the structure was that the dam had produced eleven polled calves previous to this animal and has produced one polled calf since. The presence of the horns in this one individual would indicate a possible mutation in the case under consideration. This possible mutation may have been accompanied by other changes not necessarily correlated, such as abberation, which would account for the abnormal appearance of the individual.

Only two cases are reported but both present similar characteristics. One animal was from a grade herd and the second from purebred parents. No conclusions may be drawn, but the condition may well be the result of a genetic change.

TABLE I. Width and length of pelvic regions of four dairy bulls and the animal here reported. All measurements were superficial and taken from the live animals.

	"Hybrid"	Four Bulls			
Width at tuber coxae (in.)	13.5	14.5	13.0	16.0	16.0
Width at tuber ischii (in.)	6	7.5	7.5	9.0	9.5
Length from anterior border of tuber coxae to posterior limits of tuber ischii (in.)	15.5	17.0	16.5	17.5	19.0

GENE SYMBOLS FOR USE IN COTTON GENETICS

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GENETIC work on cotton has reached a stage where the haphazard allocation of gene symbols adopted in the past has become inconvenient and confusing. We have listed the cotton genes of which descriptions are known to us and attempted to adjust their nomenclature in accordance with accepted genetic conventions and with regard to the special circumstances obtaining in the genus Gossypium.

The conventions we have adopted are as follows:

(1) Multiple allelomorph series: A series of alphabetic superscripts to a common gene symbol, e.g. the leaf shape series, L^{B}, L^{I}, L^{L}, L, l.

(2) Duplicate factors: The same gene symbol with numerical subscripts, e.g. the chlorophyll deficient duplicates, Chl_1, chl_1 : Chl_2, chl_2.

(3) Complementary factors: The same gene symbol with alphabetic subscripts, e.g. complementary crumpled, Cp_a, cp_a : Cp_b, cp_b.

A special difficulty arises in the nomenclature of cotton genes. Many characters occur in both Old World and New World groups, and frequently in the wild species also. Their genetic basis is usually similar, and in one case it has been shown that the controlling genes are homologous.[9] It is therefore desirable to have a common gene terminology throughout, but the high degree of sterility in crosses between groups makes the demonstration of homology a slow and difficult undertaking. We propose, therefore that the nomenclature of the Old World ($2n=26$) cottons be taken as the basis, and genes controlling homologous characters in the New World cultivated and Polynesian wild ($2n = 52$) and New World wild ($2n = 26$) species will be given the same symbols, but printed in ordinary type until homology has been proved, and then in italics.

Our proposals for the anthocyanin multiple allelomorph system require special explanation. The original series established in Asiatic cottons[16] consisted of six members. The two lowest members were spotless, and the four higher members, in addition to giving red petal spot, determined progressive extension of vegetative anthocyanin expression. The six members were arranged serially, R, R^{L}, R^{C}, R^{S}, r^{g}, r^{o}. Recently, however, a spotless equivalent of R^{L} has been described by Hutchinson and Ghose[22] and was given the symbol R_2^{o}. While this is satisfactory for genes now recognized, it seems to be desirable to devise a system of nomenclature which will cover any spotless types which may be found in the future. From the fact that the new spotless type is, like r^{o}, complementary with r^{g} with reference to petal spot[17, 22] it appears that there are probably at least two independent gene centres on the R protosome[18], affecting respectively petal spot and presence and distribution of anthocyanin. The probable relationship between the spotted and spotless series is shown in the table below, where the proposed symbols are indicated. The present ones are in brackets.

	Petal spotted		Spotless	
Red plant body	R_2^{RS}	(R)		
Red leaf	R_2^{LS}	(R^{L})	R_2^{LO}	(R_2^{o})
Red calyx	R_2^{CS}	(R^{C})		
Red tinged stem (i.e. basic anthocyanin gene present, expression variable but slight)	R_2^{AS}	(R^{S})	R_2^{AO}	(r^{o})
Green stem, ghost spot (basic anthocyanin gene absent)	R_2^{OS}	(r^{g})		

TABLE I. List of Recorded Genes in Cotton and their present and proposed symbols. See text for explanation of nomenclature.

Character	Old World (2n = 26) cottons			New World (2n = 52) cottons		
	Gene effect	Present symbol and authority	Proposed symbol	Gene effect	Present symbol and authority	Proposed symbol
Chlorophyll deficiency	green-chlorophyll deficient	(30)*	*Chl*	green-chlorophyll deficient duplicate	C^{ha} (11,39) C^{hb}	Chl_1 Chl_2
	green - virescent yellow	V_{11} (45)	*No alteration*	green - virescent yellow	V (26)	V
Crumpling	crumpled (complementaries)	A (16) B	Cp_a Cp_b			
Crinkled dwarf				normal-crinkled	Cr (5)	Cr
Cluster habit				normal-cluster	C^l (40)	Cl
Short fruiting branches				long-short sympodia	S^h (24)	Sh
curly leaf	curly	Cu (45)	*No alteration*			
Leaf shape	mutant broad mutant intermediate laciniated narrow broad	L^B (18) L^I L^L L l	*No alteration*	super-okra okra normal	C^s (10,32) O^o O^n	L^s L^o l
Leaf nectaries	present - absent	(27)	*Ne*			
Anthocyanin	Spotted series			Spotted series		
	red plant body	R (17)	R_1^{pp}			
	red leaf	R^L	R_2^{lp}	red leaf	R^2,R^w,R^g,(6, 9, 10, 23)†	R_2^{LW} (12)
	red calyx	R^C	R_2^{cp}			
	tinged stem	R^S	R_2^{sp}	tinged stem	S^s,^{i},^{f} (8)	$R_2^{SS,SI,SF}$
	green stem	r^g	R_2^{gp}			
	Spotless series			Spotless series		
	red leaf	R_2^O (22)	R_2^{lo}			
	tinged stem	r^p	R_2^{sp}	tinged stem	S^p (8)	R_2^{sp}
	Duplicate (ex G. anomalum)		R_1^{pp} (34)	Duplicate red spotless	R^1 (3,10,38, 40,42,44)	R_1^{pp}
	Spot reducer		*Sr* (22)			
Corolla color	yellow petal	Y (15)	Y_a	yellow	Y^p (8, 14)	Y_1
	pale	Y^p	Y_a^p	cream	y	Y_1^r
	white	y	y_a			
	pale, complementary		Y_a^p (34)			
	pale, complementary ex. G. anomalum		Y_a^p (33)			
	yellow depressor		*Ydp* (36)	yellow duplicate	Y^D (14)	Y_2
Pollen color	yellow		P_aP_b	yellow	P (7)	P
	pale (complementary)	(29)	p_a (33)			p
	cream (complementary)		p_a (36)	cream	p	
Meristic variant	increase in number of floral parts - normal	(30)	*M*			

* In this paper the authors refer to two types of chlorophyll deficiency, only one of which behave as a simple recessive. To the recessive we assign the symbol chl.

† Red leaf weak spot of Trinidad Red Leaf and red leaf extinguished spot of Cassava have both been transferred to the Sea Island background, on which they are indistinguishable. R^2R^w and R^2R^g are therefore identical and may be given the symbol R_2^{LW}.

Such symbolisation has also been attempted for the New World cottons on the basis of phenotypic appearance. The fact that members of the New World R_2 series are printed in italics indicates only that homology of the locus with that of Asiatic cottons has been established. In no case is it intended to imply identity of allelomorphs.

Below are listed the characters that have been studied genetically, the gene symbols allotted to them, and the alterations we propose. The numbers in brackets refer to the list of references attached at the end.

We are indebted to Drs. J. O. Ware and J. W. Neely of the United States Department of Agriculture, and to Mr. V. Ramanatha Ayyar of the Madras Department of Agriculture, who have criticised our manuscript and suggested a number of alterations which have been included in this list.

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TABLE I.—Continued

Character	Old World (2n = 26) cottons			New World (2n = 52) cottons		
	Gene effect	Present symbol and authority	Proposed symbol	Gene effect	Present symbol and authority	Proposed symbol
Sterility	fertile - sterile	(20)	*Stp*			
Female sterility	fertile - female sterile	Stg (41)	*No alteration*			
Petalody	normal - petalodic	p^{pd} (21)	*Ptdy*			
Boll dehiscence	more dehiscent - less dehiscent	(1)	*Dc*			
Lint colour	khaki - white	K (18)	Lo_1^K lo_1	khaki - white	K^H (13)	$Lo_1^+ - lo_1$
	khaki, duplicate		Lo_2^K (33)	duplicate	K^D (13)	$Lo_2^+ - lo_2$
	light brown	D_1 (19)	Lo_2^D (33)			
	white		lo_1			
	light brown-white, duplicate	D_2 (19)	$Lo_2^D - lo_2$ (33)			
				green - white	G^L (9)	*Lg*
Linklessness	hairy linted-glabrous linkless (complementary)	H^D (2)	H_1 (21) H_2			
	hairy linted - hairy linkless (complementary)		Li_1 (21) Li_2			
	hairy linted - hairy linkless (sometimes lethal)	h_{HL}^{L} (2)	$Li_1 - li_1$ (21)*			
Seed fuzz				naked (low lint index)-fuzzy (Upland)	N,L (3,4,25, 40,43)	*Fn*
				tufted - naked (Peruvian)	T (9,10)	*Ft*
				less fuzzy - more fuzzy (Peruvian)	F^m (22, 10)	*Fm*
	tufted - fuzzy	T (19)	*Ft*	tufted - fuzzy (Upland)	F^t (3)	*Ft*
Seed fuzz colors				green - white	G (3)	*Fgr*
				brown - white	F^r (3)	*Fbr*

* Afzal & Hutchinson (2) assigned the capital letter (H^L) to the "dominant" lethal. Hutchinson & Gadkari (21) showed that the heterozygote was strictly intermediate and the homozygote sometimes viable. On account of the close parallelism between Punjab hairy linkless and the recessive hairy linkless in *G. herbaceum* they assigned the capital letter (Li_1) to the normal gene.

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Population Trends in India

INTEREST in the approach of stationary or declining populations in the industrialized West has drawn attention from the fact that perhaps two-thirds of the world's people still live in nonindustrialized areas where population growth is controlled primarily by disease, famine, and other catastrophes. The general outlines of the situation are probably clearer in India than in any other nonindustrialized Asiatic country.

Between 1600 and 1931 the population grew from about 100 million to 353 million in the British Provinces and the Indian States. The increase between 1881 and 1931 appears to have been about 89 million, or 35 per cent, if boundary changes and the improvement of the censuses are taken into account. The percentage increase ranged from 10.6 for the United Provinces to 79.2 for Assam and 292.5 for Burma. The decade 1921-1931 was generally free from pestilence and famine with the result that the population increased nearly 11 per cent, the most rapid decennial growth at least since 1881.

Both the birth and death rates are high and fluctuate widely from area to area and year to year depending on the incidence of epidemics and of famines or severe undernutrition involved in crop failures. Incomplete and inaccurate data yield birth rates for five-year periods from 1921 to 1935 of 32 to 35, death rates of 23 to 26, and infant mortality rates of 170 to 182. The net reproduction rate, computed from estimates of age-specific fertility, is 133. Sarkar believes that the trend of both fertility and mortality is downward. However, the life tables, based on admittedly defective data, show little change in mortality.

—*Population Index,* July, 1939.

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A New Taste "Thrill"

Six (more or less) delicious flavors in one pill — depending on who eats it. Adds interest and variety to the P. T. C. "Taster Test" which has been standard for several years to demonstrate inherited differences in taste perception. Send ten cents for a sample.

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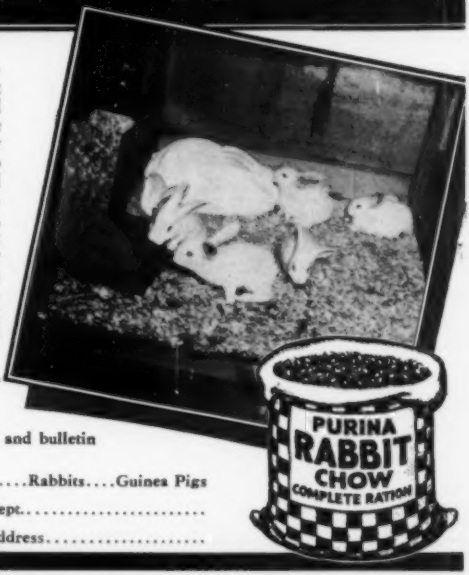

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Please send sample of diet for following animals and bulletin giving ingredients, cost and feeding directions.

I keep about (No.).....Rats.....Mice.....Dogs.....Rabbits....Guinea Pigs

Name.....Dept.....

Institution.....Address.....

Material for Demonstrating Salivary Gland Chromosomes

Interrelated demonstration material—microscopic slides, chromosome maps, lantern slides and literature—make giant chromosomes available for classroom use.

MICROSCOPIC SLIDES OF SALIVARY GLAND CHROMOSOMES

Slide of Normal Salivary Chromosomes of *D. melanogaster*, each	$2.25
Three Slides	5.00
Slide with drawing identifying all chromosomes in two figures (Female)	4.00*
Slide showing Y-chromosome (Male)	4.00
Slide showing synapsis of normal and inverted X-chromosome (loop)	4.00*
Slide showing figure of an autosomal inversion	4.00*
Slide showing synapsis of translocated and normal chromosomes	4.00*
Slide showing a small deficiency (deletion)	4.00*
Salivary chromosomes of *D. virilis*	2.25
Three Slides	5.00
Normal Somatic (ganglion) chromosomes of *D. melanogaster* *(Magnification of at least 1000 × necessary to view satisfactorily.)*	2.50
1 Slide of each of above (9 slides—6 drawings)	25.00

* Three or more slides, 1 of each kind @ $3.50 each. Three or more of same kind @ $3.00 each.

SALIVARY GLAND CHROMOSOME MAPS

Painter's cytogenetic map of the salivary chromosomes, 9½ by 18 inches, line-cut showing major chromosome details and the genes approximately located to end of 1934, mailed unfolded	$ .50
Bridges' reference map of the banding of the salivary chromosomes, 9½ by 25 inches, halftone on heavy coated paper, unfolded	1.00
Bridges' Revised Map of the X-Chromosome—9½" x 18", unfolded	.75
Folded copies of Bridges' map, on lighter paper	.35
Hughes' map of Salivary Chromosomes of *Drosophila Virilis* (9½ by 18)	.75
Dobzhansky's map of Salivary Chromosomes of *Drosophila Azteca* (9½ by 18)	.75
1 copy each of Painter's Hughes' Dobzhansky's, and Bridges' two maps (5 maps in all)	3.00

LANTERN SLIDES OF SALIVARY CHROMOSOME MATERIAL

†21-323.	Salivary chromosomes in the Nucleus	$ .75
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25-466.	Giant Chromosomes Compared with "Normal", showing relative sizes	.75
25-469.	Synapsis of Normal and Inverted Chromosomes	.75
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25-472.	Painter's Cyto-Genetic Map of Salivary Chromosomes (Insert)	.75
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26-62.	Bridges' Reference Map of the Salivary Chromosomes of Drosophila	.75
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26-178.	The Entire Salivary Gland Showing Nuclei (Sciara)	.75
26-179.	Salivary Gland Chromosomes in the Cell (Sciara)	.75
26-182.	Structure of Salivary Gland Chromosomes	.75
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27-305.	Map of Salivary Gland Chromosomes of *D. Virilis*	.75
29-12.	Bridges' Revised Map of the S. G. X-Chromosome	.75
Set of 15 Lantern Slides		9.50
Any twelve of above lantern slides		8.00

† Volume and page number of illustration in the JOURNAL OF HEREDITY.

ILLUSTRATED LITERATURE

"Symposium Reprint" on Salivary Gland Chromosomes, containing articles from the JOURNAL OF HEREDITY, 1934-1938, by Painter, Bridges, Metz, Mackensen and Hughes—four Salivary Gland Chromosome maps, 15 illustrations, 54 pages $3.00

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